

## STIC Database Tracking Number:

**To: Examiner Lena NAJARIAN**  
**Location: KNX 5A59**  
**Art Unit: 3686**  
**Date: 04/14/09**  
**Case Serial Number: 09/534946**

**From: Matthew Hogan**  
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## Search Notes

Dear Examiner NAJARIAN:

Please find attached the results of your FULL TEMPLATE search for the above-referenced case. The search was conducted in Dialog, EBSCO, and ProQuest (Financial Times).

I have listed *potential* references of interest in the first part of the search results. However, please be sure to scan through the entire report. There may be additional references that you find useful.

Please note that the results, after the potential references of interest, proceed through an Inventor search (which is provided without regard to priority date) and then to results in both Abstract and Full Text databases (which are more directly screened for priority date).

If you have any questions about the search, or need a refocus, please do not hesitate to contact me.

Thank you for using the EIC, and we look forward to your next search!

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## I. Potential References of Interest

*\* EIC-Searcher identified "potential references of interest" are selected based on the terms/concepts provided in the examiner's search request.*

2/5,K/2 (Item 1 from file: 129) Links

PHIND(Archival)

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00415100

**Coronary artery disease (CAD) study supports more aggressive diagnosis**

Clinica 618 p 24 , September 05, 1994 (19940905)

**Story Type: F Word Count: 409**

Aggressive diagnosis followed by the appropriate cholesterol lowering treatment could save millions of dollars in intervention and surgery, according to two US researchers.

metabolic manipulation beats intervention

Drs Robert Superko and Ronald Krauss of the Cholesterol Research Center at the University of California reviewed ten randomised, controlled studies involving a total of 2,095 patients, published between 1984 and 1994. As well as looking at arteriographic and clinical outcomes they compared the cost-effectiveness of the strategies with bypass surgery and angioplasty. The ten studies included non-pharmacological, single and multiple drug therapy. The ten studies "have documented that there is a cost-effective alternative to coronary bypass surgery and coronary angioplasty," says Dr Superko. The cost of metabolic manipulation, including physicians' and nursing fees, laboratory and drug costs is about \$1,300 to \$4,000 a year, depending on which drugs are used, compared with \$35,000 for CABG or \$13,000 for PTCA, he says.

If more aggressive therapy were adopted across the US, an estimated 130,000 bypass procedures could be avoided over a two-and-a-half year period - creating a real saving of \$4,600 million. However the cost difference narrows considerably for the most expensive pharmacological regime (c\$4,000 pa).

studies endorse LDL focus

"If you have heart disease and you want an aggressive programme with a good chance of achieving regression, first you need an aggressive diagnostic programme to determine why you have heart disease, so your therapy can be directed at the specific abnormality you have," says Dr Superko. The combined message from the arteriographic trials is that a more aggressive approach to lowering LDL cholesterol for secondary protection is justified.

Despite LDL values that might not place patients with documented CAD at risk, it is likely that these patients too will benefit from aggressive lipoprotein manipulation.

detailed analysis essential

Much work remains to be done to define precisely which patient subgroups will benefit most from which single or combination therapy, say the researchers. It is no longer adequate to define improvements as change in total cholesterol, LDL, HDL or triglycerides, they say. Detailed lipoprotein analysis can enhance the clinician's ability to identify correctly lipoprotein abnormality and prescribe the correct therapy that will be physiologically and economically effective. Dr Superko recommends LDL and Lp(a) phenotype determination, methionine load testing, subclass distribution for **HDL2b** (the subclass associated with most improvements), Apo E isoform characterisation, Apo B and fibrinogen determination. The cost of wide-ranging diagnostic work-up is repaid in targeted treatment and reduced clinical events, he says.

2/9,K/5 (Item 1 from file: 148) [Links](#)

Gale Group Trade & Industry DB

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08653755 **Supplier Number:** 18275566 (THIS IS THE FULL TEXT )

**Berkeley HeartLab, Inc. Gains Exclusive License to New Heart Disease Diagnostic Test Panel from Ernest Orlando Lawrence Berkeley National Laboratory; New Diagnostic Test Panel Identifies Key Risk Factors Cited by American College of Cardiology.**

Business Wire , p5131204

May 13 , 1996

**Language:** English

**Record Type:** Fulltext

**Word Count:** 1687 **Line Count:** 00144

**Text:**

SAN MATEO, Calif.--(BUSINESS WIRE)--May 13, 1996--Berkeley HeartLab, Inc., a company focusing on new diagnostic technology for detecting heart disease, announced today it has signed a \$3.8 million, five-year research agreement with a technology option with Ernest Orlando Lawrence Berkeley National Laboratory (LBNL) to acquire exclusive worldwide rights to a new diagnostic technology that is a major improvement over existing cholesterol tests in measuring heart disease risk factors.

Cardiovascular disease is the nation's leading cause of death in the U.S., accounting for 35 to 40 percent of all deaths.

The proprietary screening technology is the foundation for a new diagnostic test panel being developed by the newly formed Berkeley HeartLab, a privately-held company that will commercialize the test panel, making it widely available to the public for the first time. The new technology is seen as the next generation in heart disease screening because it can identify the large number of heart disease candidates who have the same cholesterol profile as those who do not develop heart disease. Early screening for heart disease risk will assist physicians in better selecting appropriate therapy. In many cases, customized therapy will halt progression of coronary artery disease and therefore reduce the need for expensive surgery.

#### Measures Risk Factors Cited by ACC

Berkeley HeartLab also said the new heart disease test panel measures several newly discovered risk factors and abnormalities, including the size of low-density lipoprotein particles (LDL) -- often referred to as "bad cholesterol." The new abnormalities are among the major identifiable and manageable heart disease risk factors cited by the American College of Cardiology in a task force statement published in the association's April 1996 issue of the Journal of the American College of Cardiology. As a result, Berkeley HeartLab expects that cardiologists will begin to use the new test panel as a "first-line" screening for heart disease patients and family members.

#### Next Generation of Cholesterol Testing

Cardiovascular disease ranks as the leading cause of mortality and morbidity in the United States, causing 954,000 deaths in 1993, according to the American Heart Association. Of this total, more than 489,000 deaths were due to coronary artery disease in the United States. Current testing methods used to determine an individual's risk of developing heart disease are generally confined to measuring total cholesterol, LDL, high-density lipoproteins (HDL or "good cholesterol") and triglycerides. Risk factors identified by these measurements include elevated cholesterol and elevated triglycerides, elevated LDL levels and low HDL levels. But these tests do not identify a substantial portion of patients who eventually develop heart disease.

#### Measuring New High-Risk Factors

A research team led by Ronald Krauss, M.D., director of the Department of Molecular and Nuclear Medicine at LBNL and H. Robert Superko, M.D., director of the Cholesterol, Genetics and Heart Disease Institute has developed a 10-test investigative panel which includes current parameters of cardiovascular risk -- total cholesterol, LDL, HDL and triglycerides -- plus six new tests designed to identify high-risk factors.

The 10-test panel has been used by LBNL researchers to investigate thousands of patients since 1990, as part of the lab's clinical research program. The licensing of the technology will allow the test to be made widely available commercially. Berkeley HeartLab said it plans to seek 510K approval for the investigative panel from the U.S. Food and Drug

Administration (F.D.A.)

In addition to measuring current cardiovascular risk factors, the new diagnostic technology provide six other risk factor tests, including LDL particle size:

LDL Particle Size -- Low Density Lipoprotein, or LDL, particles contribute to heart disease risk and high LDL-cholesterol is well established as a heart disease risk factor. However, most people with heart disease do NOT have classic elevated LDL cholesterol. LDL particles can be present in a variety of sizes and the small LDL's (known as LDL pattern B) create substantially higher heart disease risk compared to large LDL's (known as LDL pattern A) even when LDL cholesterol is "normal."

Measurement of LDL cholesterol does not determine these two categories of LDL. The small LDL characteristic is determined, in any given individual, by inherited traits. Approximately 50 percent of men with heart disease express the small LDL trait and approximately 50 percent of their first degree relatives can express it as well. The presence of small LDL increases the risk of heart disease by 300 percent.

Patients with LDL pattern B can be successfully treated, often with a combination of diet, weight control, and lipid lowering medications. Diet and drug therapy recommendations are different for LDL pattern A individuals. Patients with LDL pattern B can respond quite well to specialized lipid lowering therapy and achieve heart disease stability, and some degree of regression.

HDL Subclassification -- HDL participated in "reverse cholesterol transport." An abundance of **HDL2b** reflects good "reverse cholesterol transport," while a deficiency of **HDL2b** reflects poor "reverse cholesterol transport." HDL cholesterol does not reflect the proportion of the different types of HDL's. Low **HDL2b** can be improved by specific therapy.

Lp(a) - Lp(a) is an LDL particle with the protein (a) attached. This inherited trait is present in approximately 33 percent of heart disease patients and in 50 percent of their first degree relatives. It increases the risk of heart disease 300 percent and is not detected on routine blood tests. It is a powerful predictor of heart attacks in "young" men, and of vein graft blockage following bypass surgery. The presence of Lp(a) greatly increases the danger of other risk factors.

Apoprotein B - Apoprotein B is a single protein attached to the LDL particle. This test gives a more accurate measurement of the relative number of LDL particles than the current standard cholesterol blood test. Elevated apoprotein B identifies a high risk state even in the presence of "normal" LDL cholesterol. Elevated apoprotein B can be reduced with therapy.

Apoprotein AI - Apoprotein AI is one of several proteins attached to the HDL particle. It may be a better predictor of heart disease risk than HDL cholesterol. Low level of apoprotein AI can be increased with several therapies.

Apoprotein E Isoforms - Apoprotein E is attached to several lipoprotein classes and differences (isoforms) exist in "normal" and "abnormal" forms which are inherited. The presence of abnormal forms can predispose individuals to heart disease and blood lipid abnormalities. The presence, or absence, of different isoforms affects the potential success of diet and drug therapy.

The addition of these new tests provides physicians with more relevant diagnostic information for identifying heart disease candidates and assessing subsequent treatment. Early screening for and management of heart disease will allow physicians to more accurately design a customized treatment approach, reducing the need for expensive hospitalization or surgical intervention.

"The commercialization of this technology allows us to bring immediate benefits to millions of Americans who now suffer from cardiovascular disease," said Robert L. Swift, Ph.D., president and chief executive officer of Berkeley HeartLab. "More importantly, we will be able to identify the millions of Americans who have an inherited cardiovascular risk factors, so that the disease does not progress to a stage requiring costly surgical treatment."

Swift said Berkeley HeartLab will operate a centralized diagnostic service laboratory in Berkeley, Calif., that will analyze patient blood samples provided by cardiologists and a range of other physicians. The laboratory is certified under the F.D.A.'s CLIA (Clinical Laboratory Improvement Amendments) regulations. The commercial testing service is expected to begin operation in May 1996.

Researchers at the LBNL, who first isolated lipoproteins in 1949, have been world leaders in studying the connections between cholesterol and heart disease for more than 40 years. The LBNL was also the first to identify subclasses of lipoproteins and to determine that the ratio of high density-to-low density lipoproteins is a strong indicator of heart disease. The development of the testing technology and the licensing of it to the private sector is part of the laboratory's charter to convert its research breakthroughs into viable technologies for civilian applications.

#### First Product from New Company

The technology licensed from LBNL is the basis of the first product to be marketed by the newly formed Berkeley HeartLab, Inc., a privately-held company founded in March 1996. The company has raised an initial round of financing through individual investors. Berkeley HeartLab is headed by a management team that includes co-founder Robert L. Swift, Ph.D., president and chief executive officer, co-founder Dennis J. Sheehan, M.D., a member of the board of directors, and David L. Kaufman, M.D., vice president, technical operations. H. Robert Superko, an internationally known lipid specialist and director of the Cholesterol, Genetics and Heart Disease Institute, serves as medical advisor to the company.

Robert L. Swift, Ph.D., co-founder, president, chief executive officer and director, was a co-founder and former vice president of operations and development of COR Therapeutics. Prior to joining COR, Dr. Swift was director, clinical research at Genentech and director, product planning and development at Pfizer Pharmaceuticals.

Dennis J. Sheehan, M.D., co-founder and a member of the board of directors, is a fellow of the American College of Cardiology and the American Society of Angiography. He is a cardiology consultant at Sequoia Hospital, Redwood City, CA, and a clinical assistant professor of medicine at Stanford University Medical Center.

David L. Kaufman, M.D., M.P.H., vice president, technical operations, was previously vice president - clinical affairs and medical director, Circadian, Inc., where he developed and managed disease management programs and participated in development and manufacture of diagnostic and

electrosurgical devices.

CONTACT: Berkeley HeartLab, Inc.

Robert Swift, 415/378-8513

or

StratiPoint Group, Inc.

Mike Jackman or Carole Melis, 415/326-0420

5/5,K/11 (Item 11 from file: 5) [Links](#)

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Biosis Previews(R)

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11380149 **Biosis No.:** 199294081990

**REDUCED HDL-2 CHOLESTEROL SUBSPECIES AND ELEVATED POSTHEPARIN HEPATIC LIPASE ACTIVITY IN OLDER MEN WITH ABDOMINAL OBESITY AND ASYMPTOMATIC MYOCARDIAL ISCHEMIA**

**Author:** KATZEL L I (Reprint); COON P J; BUSBY M J; GOTTLIEB S O; KRAUSS R M; GOLDBERG A P

**Author Address:** BALTIMORE VA MED CENT, GERIATRICS SERV, 3900 LOCH RAVEN BOULEVARD, BALTIMORE, MD 21218, USA\*\*USA

**Journal:** Arteriosclerosis and Thrombosis 12 ( 7 ): p 814-823 1992

**ISSN:** 1049-8834

**Document Type:** Article

**Record Type:** Abstract

**Language:** ENGLISH

**Abstract:** Silent myocardial ischemia (SI), an asymptomatic manifestation of coronary artery disease (CAD), was identified in 10% of apparently healthy nonsmoking, nondiabetic older (60  $\pm$  7 years, mean  $\pm$  SD) men with normal plasma cholesterol levels. We hypothesized that in the absence of other major risk factors for CAD, the men with SI would have reduced plasma levels of high density lipoprotein (HDL) and HDL2 subspecies due to an upper-body fat distribution (waist-to-hip ratio [WHR]), hyperinsulinemia, and abnormal postheparin plasma lipoprotein lipase (LPL) and hepatic lipase (HL) activities. Compared with 47 normal control subjects of similar age, obesity, and maximal aerobic capacity, the 18 men with SI had higher plasma triglyceride (TG) (162  $\pm$  71 versus 102  $\pm$  39 mg/dl,  $p < 0.001$ ) and lower HDL-C (33  $\pm$  6 versus 37  $\pm$  7 mg/dl,  $p < 0.02$ ) levels with no difference in low density lipoprotein cholesterol level. The HDL2b and HDL2a subspecies measured by gradient gel electrophoresis were also lower in the men with SI ( $p < 0.01$ ). The plasma glucose and insulin responses during an oral glucose tolerance test were the same in both groups. Postheparin plasma HL activity was significantly higher in 12 men with SI than in 41 control subjects (34  $\pm$  8 versus 27  $\pm$  10  $\mu$ mol/ml  $\cdot$  hr<sup>-1</sup>,  $p < 0.03$ ) and was correlated with log insulin area ( $r = 0.36$ ,  $p < 0.05$ ) and WHR ( $r = 0.32$ ,  $p < 0.05$ ) in the control subjects but not in the men with SI. In the control group, the percent HDL2b subspecies was correlated inversely with postheparin plasma HL activity ( $r = -0.46$ ,  $p < 0.01$ ,  $n = 41$ ) as well as WHR ( $r = -0.49$ ,  $p < 0.001$ ,  $n = 47$ ) and log insulin area ( $r = -0.37$ ,  $p < 0.05$ ,  $n = 47$ ) but not in the men with SI. Postheparin LPL activity was the same in both groups of



men and did not correlate with HDL, WHR, insulin, or plasma TG levels. As the control subjects and men with SI had comparable degrees of abdominal obesity and hyperinsulinemia, these results suggest that the reduced HDL-C levels in men with SI may be related to elevations in HL activity. Thus, abdominal obesity, hyperinsulinemia, elevated TG levels, and low HDL-C and HDL2 subspecies level may predispose these older men to atherosclerosis.

**Registry Numbers:** 57-88-5: CHOLESTEROL; 9001-62-1: LIPASE

**Descriptors:** HIGH DENSITY LIPOPROTEIN ATHEROSCLEROSIS HYPERINSULINEMIA  
TRIGLYCERIDE LEVELS STATISTICS

**DESCRIPTORS:**

**Major Concepts:** Blood and Lymphatics--Transport and Circulation; Cardiovascular Medicine-- Human Medicine, Medical Sciences; Endocrine System--Chemical Coordination and Homeostasis; Enzymology-- Biochemistry and Molecular Biophysics; Geriatrics--Human Medicine, Medical Sciences; Nutrition; Physiology; Skeletal System--Movement and Support

**Biosystematic Names:** Hominidae--Primates, Mammalia, Vertebrata, Chordata, Animalia

**Common Taxonomic Terms:** Animals; Chordates; Humans; Mammals; Primates; Vertebrates

**Chemicals & Biochemicals:** CHOLESTEROL; LIPASE

**Concept Codes:**

04500 Mathematical biology and statistical methods

10064 Biochemistry studies - Proteins, peptides and amino acids

10066 Biochemistry studies - Lipids

10808 Enzymes - Physiological studies

11314 Chordate body regions - Abdomen

12002 Physiology - General

13203 Nutrition - Malnutrition and obesity

14506 Cardiovascular system - Heart pathology

14508 Cardiovascular system - Blood vessel pathology

15002 Blood - Blood and lymph studies

17008 Endocrine - Pancreas

18002 Bones, joints, fasciae, connective and adipose tissue - Anatomy

24500 Gerontology

**Biosystematic Codes:**

86215 Hominidae

**Abstract:** ...dl,  $p < 0.02$ ) levels with no difference in low density lipoprotein cholesterol level. The **HDL2b** and HDL2a subspecies **measured** by gradient gel electrophoresis were also lower in the men with SI ( $p < 0.01$ ...

5/5,K/25 (Item 6 from file: 73) [Links](#)

Fulltext available through: [STIC Full Text Retrieval Options](#)

EMBASE

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0072270575 **EMBASE No:** 1982013193

**Differential use of apolipoproteins and lipoproteins as discriminators for an atherogenic risk**

DIFFERENTIALEINSATZ VON APOLIPOPROTEINEN UND LIPOPROTEINEN ALS  
DISKRIMINATOREN FÜR EIN ATHEROGENES RISIKO

Mertz D.P.; Goehmann E.; Ostertag J.

Klin. am Park, 4934 Horn-Bad Meinberg, Germany

**Corresp. Author/Affil:** : Klin. am Park, 4934 Horn-Bad Meinberg, Germany

Medizinische Welt ( MED. WELT ) ( Germany ) December 1, 1981 , 32/43 (1611-1615)

**CODEN:** MEWEA **ISSN:** 0025-8512

**Document Type:** Journal ; Article **Record Type:** Abstract

**Language:** German

Apolipoproteins are now increasingly accepted as better indicators of atherosclerotic change than serum lipoproteins. Tests to verify this were carried out in 50 randomly selected patients in an ambulant practice. Lipid and lipoprotein values were collected; lipid values lose their predictive quality with age whereas lipoprotein value remains significantly associated with atherosclerotic change even in old age. As high density lipoproteins (HDL) represent a variety of substances consisting of various main and auxiliary components and because this composition can change during circulation it is suggested that **HDL2b** are more reliable in **predicting** any protective effect in serum. This however, can only be carried out by a few specialised laboratories; determination of the serum apolipoprotein concentration is a reliable indirect way of assessing HDL2a and 2b value as they appear to be narrowly connected.

**Drug Descriptors:**

\* apolipoprotein; \*lipoprotein

**Medical Descriptors:**

\* atherosclerosis; \*risk factor

cardiovascular system; major clinical study

**SECTION HEADINGS:**

Cardiovascular Diseases and Cardiovascular Surgery

Human Genetics

Clinical and Experimental Biochemistry

...and auxiliary components and because this composition can change during circulation it is suggested that **HDL2b** are more reliable in **predicting** any protective effect in serum. This however, can only be carried out by a few...

## II. Inventor Search

### A. Dialog

[File 347] **JAPIO** Dec 1976-2008/Oct(Updated 090220)

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[File 348] **EUROPEAN PATENTS** 1978-200915

(c) 2009 European Patent Office. All rights reserved.

[File 349] **PCT FULLTEXT** 1979-2009/UB=20090402IUT=20090326

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[File 350] **Derwent WPIX** 1963-2009/UD=200919

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Set	Items	Description
S1	17	S AU=(RUDERMAN, F? OR RUDERMAN F? OR SHEWMAKE D? OR SHEWMAKE, D?)
S2	17	IDPAT (sorted in duplicate/non-duplicate order)
S3	12	IDPAT (primary/non-duplicate records only)

3/3,K/1 (Item 1 from file: 350) [Links](#)

Fulltext available through: [Order File History](#)

Derwent WPIX

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0016751821 & & *Drawing available*

WPI Acc no: 2007-466893/200745

Related WPI Acc No: 2007-282358; 2007-455965; 2007-456050; 2007-466963

XRAM Acc no: C2007-169786

XRPX Acc No: N2007-354279

**Patient monitoring system comprises database for storing patient's blood test information, monitoring device monitoring the patient's vital sign and exercise information that is received by database, and Internet-based system**

Patent Assignee: BERKELEY HEARTLAB INC (BYRK-N)

Inventor: BANET M; DHILLON M; FLEMING A; HALL C; LANIER V W; **RUDERMAN F**; SCHULTZ R; **SHEWMAKE D**; VISSER H

Patent Family ( 2 patents, 115 & countries )

Patent Number	Kind	Date	Application Number	Kind	Date	Update	Type
US 20070071643	A1	20070329	US 2005721617	P	20050929	200745	B
			US 2006522589	A	20060918		
WO 2007040963	A2	20070412	WO 2006US36212	A	20060918	200745	E

Priority Applications (no., kind, date): US 2005721617 P 20050929; US 2006522589 A 20060918

Patent Details						
Patent Number	Kind	Lan	Pgs	Draw	Filing Notes	
US 20070071643	A1	EN	13	4	Related to Provisional	US 2005721617
WO 2007040963	A2	EN				
National Designated States,Original	AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HN HR HU ID IL IN IS JP KE KG KM KN KP KR KZ LA LC LK LR LS LT LU LV LY MA MD MG MK MN MW MX MY MZ NA NG NI NO NZ OM PG PH PL PT RO RS RU SC SD SE SG SK SL SM SV SY TJ TM TN TR TT TZ UA UG US UZ VC VN ZA ZM ZW					
Regional Designated States,Original	AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IS IT KE LS LT LU LV MC MW MZ NA NL OA PL PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW					

...Inventor: **RUDERMAN F**... **SHEWMAKE D** Original Publication Data by  
 AuthorityArgentina**Publication No.** ...Inventor name & address:**Shewmake, David**... **Ruderman, Frank**...  
**SHEWMAKE, David**... **RUDERMAN, Frank**

3/3,K/2 (Item 2 from file: 350) [Links](#)

Fulltext available through: [Order File History](#)

Derwent WPIX

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0016740896 & & *Drawing available*

WPI Acc no: 2007-455965/200744

Related WPI Acc No: 2007-282358; 2007-456050; 2007-466893; 2007-466963

XRAM Acc no: C2007-165775

XRPX Acc No: N2007-345308

**Low-density lipoprotein cholesterol subfraction`s particle number calculating method, involves analyzing modified distribution of particle and total low-density lipoprotein particle number value**

Patent Assignee: BERKELEY HEARTLAB (BERK-N); BERKELEY HEARTLAB INC (BYRK-N)

Inventor: BANET M; BOGGESS C; BROWNING R; CLENDENEN F; DHILLON M; FLEMING A; HALL C; LANIER V W; **RUDERMAN F**; SCHULTZ R; VISSER H; **SHEWMAKE D**

Patent Family ( 8 patents, 116 & countries )

Patent Number	Kind	Date	Application Number	Kind	Date	Update	Type
US 20070072302	A1	20070329	US 2005721617	P	20050929	200744	B
			US 2005721665	P	20050929		
			US 2005721756	P	20050929		
			US 2005721825	P	20050929		
			US 2005722051	P	20050929		
			US 2006522591	A	20060918		

WO 2007040961	A2	20070412	WO 2006US36174	A	20060918	200744	E
WO 2007040974	A2	20070412	WO 2006US36310	A	20060918	200744	E
WO 2007040975	A2	20070412	WO 2006US36311	A	20060918	200744	E
WO 2007040975	A3	20071011	WO 2006US36311	A	20060918	200768	E
WO 2007040963	A3	20071025	WO 2006US36212	A	20060918	200771	E
WO 2007040974	A3	20071101	WO 2006US36310	A	20060918	200774	E
EP 1929290	A2	20080611	EP 2006803789	A	20060918	200841	E
			WO 2006US36310	A	20060918		

Priority Applications (no., kind, date): US 2005721617 P 20050929; US 2005721665 P 20050929; US 2005721756 P 20050929; US 2005721825 P 20050929; US 2005722051 P 20050929; US 2006522591 A 20060918

#### Patent Details

Patent Number	Kind	Lan	Pgs	Draw	Filing Notes	
US 20070072302	A1	EN	10	4	Related to Provisional	US 2005721617
					Related to Provisional	US 2005721665
					Related to Provisional	US 2005721756
					Related to Provisional	US 2005721825
					Related to Provisional	US 2005722051
WO 2007040961	A2	EN				
National Designated States,Original	AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HN HR HU ID IL IN IS JP KE KG KM KN KP KR KZ LA LC LK LR LS LT LU LV LY MA MD MG MK MN MW MX MY MZ NA NG NI NO NZ OM PG PH PL PT RO RS RU SC SD SE SG SK SL SM SV SY TJ TM TN TR TT TZ UA UG US UZ VC VN ZA ZM ZW					
Regional Designated States,Original	AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IS IT KE LS LT LU LV MC MW MZ NA NL OA PL PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW					
WO 2007040974	A2	EN				
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Regional Designated States,Original	AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IS IT KE LS LT LU LV MC MW MZ NA NL OA PL PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW					
WO 2007040975	A2	EN				
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	US UZ VC VN ZA ZM ZW
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WO 2007040975	A3 EN
National Designated States,Original	AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HN HR HU ID IL IN IS JP KE KG KM KN KP KR KZ LA LC LK LR LS LT LU LV LY MA MD MG MK MN MW MX MY MZ NA NG NI NO NZ OM PG PH PL PT RO RS RU SC SD SE SG SK SL SM SV SY TJ TM TN TR TT TZ UA UG US UZ VC VN ZA ZM ZW
Regional Designated States,Original	AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IS IT KE LS LT LU LV MC MW MZ NA NL OA PL PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW
WO 2007040963	A3 EN
National Designated States,Original	AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HN HR HU ID IL IN IS JP KE KG KM KN KP KR KZ LA LC LK LR LS LT LU LV LY MA MD MG MK MN MW MX MY MZ NA NG NI NO NZ OM PG PH PL PT RO RS RU SC SD SE SG SK SL SM SV SY TJ TM TN TR TT TZ UA UG US UZ VC VN ZA ZM ZW
Regional Designated States,Original	AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IS IT KE LS LT LU LV MC MW MZ NA NL OA PL PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW
WO 2007040974	A3 EN
National Designated States,Original	AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HN HR HU ID IL IN IS JP KE KG KM KN KP KR KZ LA LC LK LR LS LT LU LV LY MA MD MG MK MN MW MX MY MZ NA NG NI NO NZ OM PG PH PL PT RO RS RU SC SD SE SG SK SL SM SV SY TJ TM TN TR TT TZ UA UG US UZ VC VN ZA ZM ZW
Regional Designated States,Original	AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IS IT KE LS LT LU LV MC MW MZ NA NL OA PL PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW
EP 1929290	A2 EN PCT Application WO 2006US36310
	Based on OPI patent WO 2007040974
Regional Designated States,Original	AL AT BA BE BG CH CY CZ DE DK EE ES FI FR GB GR HR HU IE IS IT LI LT LU LV MC MK NL PL PT RO RS SE SI SK TR

...Inventor: **RUDERMAN F...** ...**SHEWMAKE D** Original Publication Data by  
AuthorityArgentina**Publication No.** ...Inventor name & address:**RUDERMAN, Frank...** ...**Ruderman, Frank...** ...**SHEWMAKE D...** ...**RUDERMAN F...** ...**RUDERMAN, Frank...** ...**RUDERMAN F**

3/3,K/3 (Item 3 from file: 350) [Links](#)

Fulltext available through: [Order File History](#)

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0016567420 & & *Drawing available*

WPI Acc no: 2007-282358/200727

Related WPI Acc No: 2007-455965; 2007-456050; 2007-466893; 2007-466963

XRAM Acc no: C2007-103518

XRPX Acc No: N2007-208518

**Internet-based health management system for diagnosing e.g. cardiovascular disease, has monitoring device comprising data-collection component to collect glucose information and blood pressure information**

Patent Assignee: BERKELEY HEARTLAB (BERK-N); BERKELEY HEARTLAB INC (BYRK-N)

Inventor: BANET M; DHILLON M; FLEMING A; HALL C; LANIER V; LANIER V W; RUDERMAN F; SCHULTZ R; SHEWMAKE D; VISSER H

Patent Family ( 3 patents, 115 & countries )

Patent Number	Kind	Date	Application Number	Kind	Date	Update	Type
US 20070068539	A1	20070329	US 2005721825	P	20050929	200727	B
			US 2006522650	A	20060918		
WO 2007040971	A2	20070412	WO 2006US36301	A	20060918	200727	E
WO 2007040971	A3	20071011	WO 2006US36301	A	20060918	200768	E

Priority Applications (no., kind, date): US 2005721825 P 20050929; US 2006522650 A 20060918

Patent Details

Patent Number	Kind	Lan	Pgs	Draw	Filing Notes	
US 20070068539	A1	EN	10	4	Related to Provisional	US 2005721825
WO 2007040971	A2	EN				
National Designated States,Original	AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HN HR HU ID IL IN IS JP KE KG KM KN KP KR KZ LA LC LK LR LS LT LU LV LY MA MD MG MK MN MW MX MY MZ NA NG NI NO NZ OM PG PH PL PT RO RS RU SC SD SE SG SK SL SM SV SY TJ TM TN TR TT TZ UA UG US UZ VC VN ZA ZM ZW					
Regional Designated States,Original	AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IS IT KE LS LT LU LV MC MW MZ NA NL OA PL PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW					
WO 2007040971	A3	EN				
National Designated States,Original	AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HN HR HU ID IL IN IS JP KE KG KM KN KP KR KZ LA LC LK LR LS LT LU LV LY MA MD MG MK MN MW MX MY MZ NA NG NI NO NZ OM PG PH PL PT RO RS RU SC SD SE SG SK SL SM SV SY TJ TM TN TR TT TZ UA UG US UZ VC VN ZA ZM ZW					
Regional Designated States,Original	AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IS IT KE LS LT LU LV MC MW MZ NA NL OA PL PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW					

...Inventor: **RUDERMAN F... SHEWMAKE D** Original Publication Data by AuthorityArgentina**Publication No.** ...Inventor name & address:**Shewmake, David... Ruderman, Frank... SHEWMAKE, David... RUDERMAN, Frank... SHEWMAKE D... RUDERMAN F**

3/3,K/4 (Item 4 from file: 350) [Links](#)

Fulltext available through: [Order File History](#)

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0014752351

WPI Acc no: 2005-099982/200511

Related WPI Acc No: 2007-690318

XRAM Acc no: C2005-033441

XRFX Acc No: N2005-086833

**Selecting an optimal diet and exercise regimen and treating patient with cardiovascular disease involves considering low-density lipoprotein or high-density lipoprotein subclass levels and selecting diet and exercise regimen**

Patent Assignee: BERKELEY HEARTLAB INC (BYRK-N)

Inventor: ROBERTS A C; **SHEWMAKE D T**

Patent Family ( 2 patents, 1 & countries )

Patent Number	Kind	Date	Application Number	Kind	Date	Update	Type
US 20050009193	A1	20050113	US 2003473715	P	20030527	200511	B
			US 2004854402	A	20040526		
US 7226792	B2	20070605	US 2004854402	A	20040526	200737	E

Priority Applications (no., kind, date): US 2003473715 P 20030527; US 2004854402 A 20040526

Patent Details

Patent Number	Kind	Lan	Pgs	Draw	Filing Notes	
US 20050009193	A1	EN	10	0	Related to Provisional	US 2003473715

...Inventor: **SHEWMAKE D T** Original Publication Data by AuthorityArgentina**Publication No.** ...Inventor name & address:**Shewmake, David T... Shewmake, David T**

3/3,K/5 (Item 5 from file: 350) [Links](#)

Fulltext available through: [Order File History](#)

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0013997653

WPI Acc no: 2004-178837/200417



Related WPI Acc No: 2003-903193

XRAM Acc no: C2004-070819

XRPX Acc No: N2004-142161

**Normal national cholesterol education program lipid levels identification for identifying patients to be treated/monitored for cardiovascular disease, by measuring LDL/HDL particle subclass levels and identifying abnormal levels**

Patent Assignee: BERKELEY HEARTLAB INC (BYRK-N)

Inventor: BOGGESS C; CLENDENEN F; **RUDERMAN F; SHEWMAKE D**

Patent Family ( 1 patents, 1 & countries )

Patent Number	Kind	Date	Application Number	Kind	Date	Update	Type
US 20030235918	A1	20031225	US 2002390976	P	20020624	200417	B
			US 2003412838	A	20030412		

Priority Applications (no., kind, date): US 2002390976 P 20020624; US 2003412838 A 20030412

Patent Details

Patent Number	Kind	Lan	Pgs	Draw	Filing Notes	
US 20030235918	A1	EN	33	24	Related to Provisional	US 2002390976

...Inventor: **RUDERMAN F...** ...**SHEWMAKE D** Original Publication Data by

AuthorityArgentina**Publication No.** Inventor name & address:**Shewmake, David...** ...**Ruderman, Frank**

3/3,K/6 (Item 6 from file: 350) [Links](#)

Fulltext available through: [Order File History](#)

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0013802968

WPI Acc no: 2003-903193/200382

Related WPI Acc No: 2004-178837

XRAM Acc no: C2003-256666

XRPX Acc No: N2003-721236

**Method for identifying patient with normal national cholesterol education program lipid in the treatment of cardiovascular disease involves measuring low density lipoprotein or high density lipoprotein particle subclass levels**

Patent Assignee: BERKELEY HEARTLAB INC (BYRK-N)

Inventor: BOGGESS C; CLENDENEN F; **RUDERMAN F; SHEWMAKE D; SUPERKO H R**

Patent Family ( 7 patents, 100 & countries )

Patent Number	Kind	Date	Application Number	Kind	Date	Update	Type
WO 2003087844	A1	20031023	WO 2003US11375	A	20030411	200382	B
US 20030194812	A1	20031016	US 2002122081	A	20020412	200382	E
US 20040043496	A1	20040304	US 2002390796	P	20020621	200417	E
			US 2003463664	A	20030616		
AU 2003221918	A1	20031027	AU 2003221918	A	20030411	200436	E

US 6812033	B2	20041102	US 2002122081	A	20020412	200472	E
US 20050042761	A1	20050224	US 2002122081	A	20020412	200515	E
			US 2004959656	A	20041006		
US 7416895	B2	20080826	US 2002390796	P	20020621	200857	E
			US 2003412838	A	20030412		

Priority Applications (no., kind, date): US 2002122081 A 20020412; US 2002390796 P 20020621; US 2003412838 A 20030412; US 2003463664 A 20030616; US 2004959656 A 20041006

Patent Details							
Patent Number	Kind	Lan	Pgs	Draw	Filing Notes		
WO 2003087844	A1	EN	25	24			
National Designated States,Original	AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ OM PH PL PT RO RU SC SD SE SG SK SL TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW						
Regional Designated States,Original	AT BE BG CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NL OA PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW						
US 20040043496	A1	EN			Related to Provisional	US 2002390796	
AU 2003221918	A1	EN			Based on OPI patent	WO 2003087844	
US 20050042761	A1	EN			Continuation of application	US 2002122081	
					Continuation of patent	US 6812033	
US 7416895	B2	EN			Related to Provisional	US 2002390796	

...Inventor: **RUDERMAN F...** **SHEWMAKE D** Original Publication Data by AuthorityArgentina**Publication No.** Inventor name & address:**SHEWMAKE D...** **RUDERMAN F...** **Shewmake, David...** **Ruderman, Frank...** **Shewmake, David...** **Ruderman, Frank...** **Shewmake, David...** **Ruderman, Frank...** **Shewmake, David...** **Ruderman, Frank...** **SHEWMAKE, David...** **RUDERMAN, Frank**

3/3,K/7 (Item 7 from file: 350) [Links](#)

Fulltext available through: [Order File History](#)

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0010977989 & & Drawing available

WPI Acc no: 2001-602239/200168

XRPX Acc No: N2001-449418

**Cardiovascular healthcare management system for network based health diagnosis, allows physician to diagnose disease by accessing patient's personal data and test results in database, to output management information**

Patent Assignee: BERKELEY HEARTLAB INC (BYRK-N); RUDERMAN F R (RUDE-I); SHEWMAKE

D T (SHEW-D)

Inventor: **RUDERMAN F R; SHEWMAKE D T**

Patent Family ( 3 patents, 92 & countries )

Patent Number	Kind	Date	Application Number	Kind	Date	Update	Type
WO 2001041037	A2	20010607	WO 2000US32833	A	20001201	200168	B
AU 200118143	A	20010612	AU 200118143	A	20001201	200168	E
US 20030208108	A1	20031106	WO 2000US32833	A	20001201	200374	E
			US 2003169648	A	20030121		

Priority Applications (no., kind, date): US 1999168354 P 19991201; US 2000534946 A 20000324; US 2003169648 A 20030121

Patent Details

Patent Number	Kind	Lan	Pgs	Draw	Filing Notes	
WO 2001041037	A2	EN	56	32		
National Designated States,Original	AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW					
Regional Designated States,Original	AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TR TZ UG ZW					
AU 200118143	A	EN			Based on OPI patent	WO 2001041037
US 20030208108	A1	EN			PCT Application	WO 2000US32833

Inventor: **RUDERMAN F R... ..SHEWMAKE D T** Original Publication Data by  
AuthorityArgentina**Publication No.** Inventor name & address:**Shewmake, David T... ..Ruderman, Frank R... ..RUDERMAN, Frank, R... ..SHEWMAKE, David, T**

3/3K/8 (Item 8 from file: 349) [Links](#)

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01497980

**INTERNET-BASED HEALTH MANAGEMENT SYSTEM FOR IDENTIFYING AND MINIMIZING RISK FACTORS CONTRIBUTING TO METABOLIC SYNDROME**

**SYSTEME DE GESTION DE LA SANTE PAR INTERNET PERMETTANT D'IDENTIFIER ET DE MINIMISER LES FACTEURS DE RISQUE CONTRIBUANT AU SYNDROME METABOLIQUE**

**Patent Applicant/Patent Assignee:**

• **BERKELEY HEARTLAB**

839 Mitten Road, Burlingame, CA 94010; US; US (Residence); US (Nationality); (For all designated states except: US)

**Patent Applicant/Inventor:**

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- **LANIER Vance**  
3930 24th Street #12, San Francisco, CA 94114; US; US (Residence); US (Nationality); (Designated only for: US)
- **SHEWMAKE David**  
**419 Baden Street, San Francisco, CA 94131; US; US (Residence); US (Nationality); (Designated only for: US)**
- **RUDERMAN Frank**  
71 Creekside Drive, San Rafael, CA 94903; US; US (Residence); US (Nationality); (Designated only for: US)
- **BANET Matthew**  
12719 Via Felino, Del Mar, CA 92014; US; US (Residence); US (Nationality); (Designated only for: US)
- **SCHULTZ Randon**  
2519 Strongs Drive, Venice, CA 90291; US; US (Residence); US (Nationality); (Designated only for: US)
- **DHILLON Marshal**  
4580 Huggins Street, San Diego, CA 92122; US; US (Residence); US (Nationality); (Designated only for: US)
- **FLEMING Adam**  
5679 Arbor Grove Ct. #B1, San Diego, CA 92121; US; US (Residence); US (Nationality); (Designated only for: US)
- **VISSER Henk**  
8540 Costa Verde Boulevard #4425, San Diego, CA 92121; US; US (Residence); US (Nationality); (Designated only for: US)
- **...Designated only for: US)**
- **SHEWMAKE David... ...Designated only for: US)**
- **RUDERMAN Frank...**

**Legal Representative:**

- **MCDONNELL John J(agent)**  
MCDONNELL BOEHNEN HULBERT & BERGHOFF LLP, SUITE 3100, 300 South Wacker Drive, Chicago, IL 60606; US;

	Country	Number	Kind	Date
Patent	WO	200740971	A2-A3	20070412
Application	WO	2006US36301		20060918

Priorities	US	2005721825		20050929
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**Designated States:** (All protection types applied unless otherwise stated - for applications 2004+)

AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG;  
BR; BW; BY; BZ; CA; CH; CN; CO; CR; CU;  
CZ; DE; DK; DM; DZ; EC; EE; EG; ES; FI;  
GB; GD; GE; GH; GM; HN; HR; HU; ID; IL;  
IN; IS; JP; KE; KG; KM; KN; KP; KR; KZ;  
LA; LC; LK; LR; LS; LT; LU; LV; LY; MA;  
MD; MG; MK; MN; MW; MX; MY; MZ; NA; NG;  
NI; NO; NZ; OM; PG; PH; PL; PT; RO; RS;  
RU; SC; SD; SE; SG; SK; SL; SM; SV; SY;  
TJ; TM; TN; TR; TT; TZ; UA; UG; US; UZ;  
VC; VN; ZA; ZM; ZW;

[EP] AT; BE; BG; CH; CY; CZ; DE; DK; EE; ES;  
FI; FR; GB; GR; HU; IE; IS; IT; LT; LU;  
LV; MC; NL; PL; PT; RO; SE; SI; SK; TR;

[OA] BF; BJ; CF; CG; CI; CM; GA; GN; GQ; GW;  
ML; MR; NE; SN; TD; TG;

[AP] BW; GH; GM; KE; LS; MW; MZ; NA; SD; SL;  
SZ; TZ; UG; ZM; ZW;

[EA] AM; AZ; BY; KG; KZ; MD; RU; TJ; TM;

Publication Language: English

Filing Language: English

Fulltext word count: 4200

3/3K/9 (Item 9 from file: 349) [Links](#)

Fulltext available through: [Order File History](#)

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01496720

**INTERNET-BASED SYSTEM FOR MONITORING BLOOD TEST, VITAL SIGN, AND EXERCISE  
INFORMATION FROM A PATIENT**

**SYSTEME BASE SUR INTERNET POUR LE SURVEILLANCE DE TEST SANGUIN, DE SIGNES  
VITAUX ET D'INFORMATIONS D'EXERCICE D'UN PATIENT**

**Patent Applicant/Patent Assignee:**

- **BERKELEY HEARTLAB INC**

839 Mitten Road, Burlingame, CA 94010; US; US (Residence); US (Nationality); (For all designated states except: US)

**Patent Applicant/Inventor:**

- **HALL Christopher**

810 Douglass Street, San Francisco, CA 94114; US; US (Residence); US (Nationality); (Designated only for: US)

- **LANIER Vance Whitson Jr**

3930 24th Street, #12, San Francisco, CA 94114; US; US (Residence); US (Nationality); (Designated only for: US)

- **SHEWMAKE David**

**419 Baden Street, San Francisco, CA 94131; US; US (Residence); US (Nationality); (Designated only for: US)**

- **RUDERMAN Frank**

71 Creekside Drive, San Rafael, CA 94903; US; US (Residence); US (Nationality); (Designated only for: US)

- **BANET Matthew**

12719 Via Felino, Del Mar, CA 92014; US; US (Residence); US (Nationality); (Designated only for: US)

- **SCHULTZ Randon**

2519 Strongs Drive, Venice, CA 90291; US; US (Residence); US (Nationality); (Designated only for: US)

- **DHILLON Marshal**

4580 Huggins Street, San Diego, CA 92122; US; US (Residence); US (Nationality); (Designated only for: US)

- **FLEMING Adam**

5679 Arbor Grove Ct., #B1, San Diego, CA 92121; US; US (Residence); US (Nationality); (Designated only for: US)

- **VISSER Henk**

8540 Costa Verde Boulevard, #4425, San Diego, CA 92121; US; US (Residence); US (Nationality); (Designated only for: US)

- **...Designated only for: US)**

- **SHEWMAKE David... ...Designated only for: US)**

- **RUDERMAN Frank...**

**Legal Representative:**

- **MCDONNELL John J(agent)**

MCDONNELL BOEHNEN HULBERT & BERGHOFF LLP, 300 South Wacker Drive, Suite 3200, Chicago, IL 60606; US;

	Country	Number	Kind	Date
Patent	WO	200740963	A2-A3	20070412
Application	WO	2006US36212		20060918
Priorities	US	2005721617		20050929

**Designated States:** (All protection types applied unless otherwise stated - for applications 2004+)

AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG;  
BR; BW; BY; BZ; CA; CH; CN; CO; CR; CU;  
CZ; DE; DK; DM; DZ; EC; EE; EG; ES; FI;  
GB; GD; GE; GH; GM; HN; HR; HU; ID; IL;  
IN; IS; JP; KE; KG; KM; KN; KP; KR; KZ;  
LA; LC; LK; LR; LS; LT; LU; LV; LY; MA;  
MD; MG; MK; MN; MW; MX; MY; MZ; NA; NG;  
NI; NO; NZ; OM; PG; PH; PL; PT; RO; RS;  
RU; SC; SD; SE; SG; SK; SL; SM; SV; SY;  
TJ; TM; TN; TR; TT; TZ; UA; UG; US; UZ;  
VC; VN; ZA; ZM; ZW;

[EP] AT; BE; BG; CH; CY; CZ; DE; DK; EE; ES;  
FI; FR; GB; GR; HU; IE; IS; IT; LT; LU;  
LV; MC; NL; PL; PT; RO; SE; SI; SK; TR;

[OA] BF; BJ; CF; CG; CI; CM; GA; GN; GQ; GW;  
ML; MR; NE; SN; TD; TG;

[AP] BW; GH; GM; KE; LS; MW; MZ; NA; SD; SL;  
SZ; TZ; UG; ZM; ZW;

[EA] AM; AZ; BY; KG; KZ; MD; RU; TJ; TM;

Publication Language: English

Filing Language: English

Fulltext word count: 6183

3/3K/10 (Item 10 from file: 349) [Links](#)

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01496077

**METHOD FOR QUANTITATIVELY DETERMINING THE LDL PARTICLE NUMBER IN A DISTRIBUTION OF LDL CHOLESTEROL SUBFRACTIONS**

PROCEDE PERMETTANT DE DETERMINER DE MANIERE QUANTITATIVE LE NOMBRE DE PARTICULES LDL DANS UNE DISTRIBUTION DE SOUS-FRACTIONS DE CHOLESTEROL LDL

**Patent Applicant/Patent Assignee:**

- **BERKELEY HEARTLAB**

839 Mitten Road, Burlingame, CA 94010; US; US (Residence); US (Nationality); (For all designated states except: US)

**Patent Applicant/Inventor:**

- **CLENDENEN Faith**

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- **BOGGESS Christopher**

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- **RUDERMAN Frank**

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- ...Designated only for: US)

- **RUDERMAN Frank...**

**Legal Representative:**

- **MCDONNELL John J(agent)**

MCDONNELL BOEHNEN HULBERT & BERGHOFF LLP, SUITE 3100, 300 South Wacker Drive, Chicago, IL 60606; US;

	Country	Number	Kind	Date
Patent	WO	200740974	A2-A3	20070412
Application	WO	2006US36310		20060918
Priorities	US	2005722051		20050929
	US	2005721825		20050929
	US	2005721665		20050929
	US	2005721756		20050929
	US	2005721617		20050929

**Designated States:** (All protection types applied unless otherwise stated - for applications 2004+)

AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG;  
 BR; BW; BY; BZ; CA; CH; CN; CO; CR; CU;  
 CZ; DE; DK; DM; DZ; EC; EE; EG; ES; FI;  
 GB; GD; GE; GH; GM; HN; HR; HU; ID; IL;  
 IN; IS; JP; KE; KG; KM; KN; KP; KR; KZ;  
 LA; LC; LK; LR; LS; LT; LU; LV; LY; MA;  
 MD; MG; MK; MN; MW; MX; MY; MZ; NA; NG;  
 NI; NO; NZ; OM; PG; PH; PL; PT; RO; RS;  
 RU; SC; SD; SE; SG; SK; SL; SM; SV; SY;  
 TJ; TM; TN; TR; TT; TZ; UA; UG; US; UZ;  
 VC; VN; ZA; ZM; ZW;



[EP] AT; BE; BG; CH; CY; CZ; DE; DK; EE; ES;  
FI; FR; GB; GR; HU; IE; IS; IT; LT; LU;  
LV; MC; NL; PL; PT; RO; SE; SI; SK; TR;

[OA] BF; BJ; CF; CG; CI; CM; GA; GN; GQ; GW;  
ML; MR; NE; SN; TD; TG;

[AP] BW; GH; GM; KE; LS; MW; MZ; NA; SD; SL;  
SZ; TZ; UG; ZM; ZW;

[EA] AM; AZ; BY; KG; KZ; MD; RU; TJ; TM;

Publication Language: English

Filing Language: English

Fulltext word count: 4753

3/3K/11 (Item 11 from file: 349) [Links](#)

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01059072

**A METHOD FOR IDENTIFYING AT RISK CARDIOVASCULAR DISEASE PATIENTS**  
PROCEDE PERMETTANT D'IDENTIFIER DES PATIENTS PRESENTANT DES RISQUES DE  
MALADIES CARDIO-VASCULAIRES

**Patent Applicant/Patent Assignee:**

• **BERKELEY HEARTLAB INC**

839 Mitten Road, Burlingame, CA 94010; US; US(Residence); US(Nationality); (For all designated states except: US)

**Patent Applicant/Inventor:**

• **SHEWMAKE David**

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(Designated only for: US)

• **RUDERMAN Frank**

248 Bayview Drive, San Carlos, CA 94070; US; US(Residence); US(Nationality); (Designated only for: US)

• **BOGGESE Christopher**

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(Designated only for: US)

• **CLENDENEN Faith**

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• **SHEWMAKE David... ...Designated only for: US)**

• **RUDERMAN Frank...**

**Legal Representative:**

• **MCDONNELL John J(agent)**

McDonnell Boehnen Hulbert & Berghoff, 300 South Wacker, Suite 3200, Chicago, IL 60606; US;

	Country	Number	Kind	Date
Patent	WO	200387844	A1	20031023
Application	WO	2003US11375		20030411
Priorities	US	2002122081		20020412
	US	2002390796		20020621

**Designated States:** (Protection type is "Patent" unless otherwise stated - for applications prior to 2004)

AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG,  
BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ,  
DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD,  
GE, GH, GM, HR, HU, ID, IL, IN, IS, JP,  
KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT,  
LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ,  
NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD,  
SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ,  
UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

[EP] AT; BE; BG; CH; CY; CZ; DE; DK; EE; ES;  
FI; FR; GB; GR; HU; IE; IT; LU; MC; NL;  
PT; RO; SE; SI; SK; TR;

[OA] BF; BJ; CF; CG; CI; CM; GA; GN; GQ; GW;  
ML; MR; NE; SN; TD; TG;

[AP] GH; GM; KE; LS; MW; MZ; SD; SL; SZ; TZ;  
UG; ZM; ZW;

[EA] AM; AZ; BY; KG; KZ; MD; RU; TJ; TM;

Publication Language: English

Filing Language: English

Fulltext word count: 5321

3/3K/12 (Item 12 from file: 349) [Links](#)

Fulltext available through: [Order File History](#)  
PCT FULLTEXT

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00808349

**CARDIOVASCULAR HEALTHCARE MANAGEMENT SYSTEM AND METHOD**  
**PROCEDE ET SYSTEME DE GESTION DES SOINS DE SANTE CARDIOVASCULAIRES**

**Patent Applicant/Patent Assignee:**

• **BERKELEY HEARTLAB INC**

1875 South Grant St., Suite #700, San Mateo, CA 94402; US; US(Residence); US(Nationality); (For all designated states except: US)

**Patent Applicant/Inventor:**

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419 Baden Street, San Francisco, CA 94131-2831; US; US(Residence); US(Nationality); (Designated only for: US)

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• **SHEWMAKE David T...**

**Legal Representative:**

• **MCDONNELL John J(agent)**

McDonnell Boehnen Hulbert & Berghoff, 32nd Floor, 300 South Wacker Drive, Chicago, IL 60606; US;

	Country	Number	Kind	Date
Patent	WO	200141037	A2-A3	20010607
Application	WO	2000US32833		20001201
Priorities	US	99168354		19991201
	US	2000534946		20000324

**Designated States:** (Protection type is "Patent" unless otherwise stated - for applications prior to 2004)

AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG,  
BR, BY, BZ, CA, CH, CN, CR, CU, CZ, DE,  
DK, DM, DZ, EE, ES, FI, GB, GD, GE, GH,  
GM, HR, HU, ID, IL, IN, IS, JP, KE, KG,  
KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV,  
MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ,  
PL, PT, RO, RU, SD, SE, SG, SI, SK, SL,  
TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN,  
YU, ZA, ZW

[EP] AT; BE; CH; CY; DE; DK; ES; FI; FR; GB;  
GR; IE; IT; LU; MC; NL; PT; SE; TR;

[OA] BF; BJ; CF; CG; CI; CM; GA; GN; GW; ML;  
MR; NE; SN; TD; TG;

[AP] GH; GM; KE; LS; MW; MZ; SD; SL; SZ; TZ;  
UG; ZW;

[EA] AM; AZ; BY; KG; KZ; MD; RU; TJ; TM;

Publication Language: English

Filing Language: English

Fulltext word count: 8871

[File 5] **Biosis Previews(R)** 1926-2009/Apr W1  
(c) 2009 The Thomson Corporation. All rights reserved.

[File 73] **EMBASE** 1974-2009/Apr 09  
(c) 2009 Elsevier B.V. All rights reserved.

[File 155] **MEDLINE(R)** 1950-2009/Apr 13  
(c) format only 2009 Dialog. All rights reserved.

[File 34] **SciSearch(R) Cited Ref Sci** 1990-2009/Apr W1  
(c) 2009 The Thomson Corp. All rights reserved.

[File 434] **SciSearch(R) Cited Ref Sci** 1974-1989/Dec  
(c) 2006 The Thomson Corp. All rights reserved.

[File 74] **Int.Pharm.Abs** 1970-2009/Jan B1  
(c) 2009 The Thomson Corporation. All rights reserved.

[File 42] **Pharm. News Index** 1974-2009/Mar W3  
(c) 2009 ProQuest Info&Learning. All rights reserved.

[File 35] **Dissertation Abs Online** 1861-2009/Mar  
(c) 2009 ProQuest Info&Learning. All rights reserved.

[File 583] **Gale Group Globalbase(TM)** 1986-2002/Dec 13  
(c) 2002 Gale/Cengage. All rights reserved.  
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[File 65] **Inside Conferences** 1993-2009/Apr 09  
(c) 2009 BLDSC all rts. reserv. All rights reserved.

[File 2] **INSPEC** 1898-2009/Apr W1  
(c) 2009 Institution of Electrical Engineers. All rights reserved.

[File 474] **New York Times Abs** 1969-2009/Apr 13  
(c) 2009 The New York Times. All rights reserved.

[File 475] **Wall Street Journal Abs** 1973-2009/Apr 13  
(c) 2009 The New York Times. All rights reserved.

[File 99] **Wilson Appl. Sci & Tech Abs** 1983-2009/Feb  
(c) 2009 The HW Wilson Co. All rights reserved.

[File 256] **TecInfoSource** 82-2009/Dec  
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[File 15] **ABI/Inform(R)** 1971-2009/Apr 11  
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[File 9] **Business & Industry(R)** Jul/1994-2009/Apr 11  
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[File 20] **Dialog Global Reporter** 1997-2009/Apr 14  
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[File 149] **TGG Health&Wellness DB(SM)** 1976-2009/Mar W3  
(c) 2009 Gale/Cengage. All rights reserved.

[File 444] **New England Journal of Med.** 1985-2009/Dec W4  
(c) 2009 Mass. Med. Soc. All rights reserved.

[File 455] **Drug News & Perspectives** 1992-2005/Aug  
(c) 2005 Prous Science. All rights reserved.  
*\*File 455: This file is closed. Please see HELP NEWS 455 for more information.*

[File 129] **PHIND(Archival)** 1980-2009/Feb W3  
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[File 130] **PHIND(Daily & Current)** 2009/Apr 14  
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*\*File 130: Due to the Easter holiday, there were no updates to the file on 4/10 or 4/13.*

Set	Items	Description
S1	34	S AU=(RUDERMAN, F? OR RUDERMAN F? OR SHEWMAKE D? OR SHEWMAKE, D?)
S2	1	S S1 AND CHOLESTEROL?

2/5,K/1 (Item 1 from file: 5) [Links](#)  
Biosis Previews(R)  
(c) 2009 The Thomson Corporation. All rights reserved.  
18084849 **Biosis No.:** 200400466078  
**Method for identifying risk cardiovascular disease patients**

**Author:** Shewmake David (Reprint); Ruderman Frank; Boggess Christopher  
**Author Address:** San Francisco, CA, USA\*\*USA  
**Journal:** Official Gazette of the United States Patent and Trademark Office Patents 1288 ( 1 ): Nov. 2, 2004 2004  
**Medium:** e-file  
**Patent Number:** US 6812033 **Patent Date Granted:** November 02, 2004 20041102 **Patent Classification:** 436-71 **Patent Assignee:** Berkeley HeartLab, Inc., San Mateo, CA, USA **Patent Country:** USA  
**ISSN:** 0098-1133 \_(ISSN print)  
**Document Type:** Patent  
**Record Type:** Abstract  
**Language:** English

**Abstract:** The invention provides a method for identifying patients with normal NCEP lipid levels who are in need of treatment for cardiovascular disease comprising measuring one or more LDL or HDL particle subclass levels and identifying abnormal LDL or HDL subclass levels.

## DESCRIPTORS:

**Major Concepts:** Cardiovascular Medicine--Human Medicine, Medical Sciences; Clinical Chemistry--Allied Medical Sciences; Methods and Techniques

**Diseases:** cardiovascular disease--heart disease, vascular disease, diagnosis

**Mesh Terms:** Cardiovascular Diseases (MeSH)

**Chemicals & Biochemicals:** HDL {high density lipoprotein}--subclass levels; LDL {low density lipoprotein}--subclass levels

**Methods & Equipment:** risk assessment method--clinical techniques, diagnostic techniques

**Miscellaneous Terms: Concept Codes:** National **Cholesterol** Education Program {NCEP}

**Concept Codes:**

10006 Clinical biochemistry - General methods and applications

10064 Biochemistry studies - Proteins, peptides and amino acids

10066 Biochemistry studies - Lipids

12504 Pathology - Diagnostic

14506 Cardiovascular system - Heart pathology

14508 Cardiovascular system - Blood vessel pathology

**Author: Shewmake David... ..Ruderman Frank**

**DESCRIPTORS:**

**Miscellaneous Terms: Concept Codes:** National **Cholesterol** Education Program {NCEP}

### III. Text Search Results from Dialog (Full Text dbs)

#### A. Full-Text Databases – PATENT

[File 348] **EUROPEAN PATENTS** 1978-200915

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[File 349] **PCT FULLTEXT** 1979-2009/UB=20090402|UT=20090326

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Set	Items	Description
S1	38	S HDL()2B OR HDL2B OR (HIGH(2W)LIPOPROTEIN?) (2N) (2B OR 2()B) OR H()D()L() (2()B OR 2B) OR HDL2()B
S2	0	S S1 NOT AY>1999



## B. Full-Text Databases – NON-PATENT

[File 15] **ABI/Inform(R)** 1971-2009/Apr 11

(c) 2009 ProQuest Info&Learning. All rights reserved.

[File 9] **Business & Industry(R)** Jul/1994-2009/Apr 11

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[File 810] **Business Wire** 1986-1999/Feb 28

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[File 624] **McGraw-Hill Publications** 1985-2009/Apr 14

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[File 634] **San Jose Mercury** Jun 1985-2009/Apr 10

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[File 20] **Dialog Global Reporter** 1997-2009/Apr 14

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Set	Items	Description
S1	26	S HDL()2B OR HDL2B OR (HIGH(2W)LIPOPROTEIN?) (2N) (2B OR 2()B) OR H()D()L() (2()B OR 2B) OR HDL2()B
S2	6	S S1 NOT PY>1999

[File 149] **TGG Health&Wellness DB(SM)** 1976-2009/Mar W3  
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Set	Items	Description
S1	10	S HDL()2B OR HDL2B OR (HIGH(2W)LIPOPROTEIN?) (2N) (2B OR 2()B) OR H()D()L() (2()B OR 2B) OR HDL2()B
S2	2	S S1 NOT PY>1999

2/5,K/2 (Item 1 from file: 129) [Links](#)  
PHIND(Archival)  
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00415100

### **Coronary artery disease (CAD) study supports more aggressive diagnosis**

Clinica 618 p 24 , September 05, 1994 (19940905)  
**Story Type: F Word Count: 409**

Aggressive diagnosis followed by the appropriate cholesterol lowering treatment could save millions of dollars in intervention and surgery, according to two US researchers. metabolic manipulation beats intervention  
Drs Robert Superko and Ronald Krauss of the Cholesterol Research Center at the University of California reviewed ten randomised, controlled studies involving a total of 2,095 patients, published between 1984 and 1994. As well as looking at arteriographic and clinical outcomes they compared the cost-effectiveness of the strategies with bypass surgery and angioplasty. The ten studies included non-pharmacological, single and multiple drug therapy. The ten studies "have documented that there is a cost-effective alternative to coronary bypass surgery and coronary angioplasty,"

says Dr Superko. The cost of metabolic manipulation, including physicians' and nursing fees, laboratory and drug costs is about \$1,300 to \$4,000 a year, depending on which drugs are used, compared with \$35,000 for CABG or \$13,000 for PTCA, he says. If more aggressive therapy were adopted across the US, an estimated 130,000 bypass procedures could be avoided over a two-and-a-half year period - creating a real saving of \$4,600 million. However the cost difference narrows considerably for the most expensive pharmacological regime (c\$4,000 pa). studies endorse LDL focus

"If you have heart disease and you want an aggressive programme with a good chance of achieving regression, first you need an aggressive diagnostic programme to determine why you have heart disease, so your therapy can be directed at the specific abnormality you have," says Dr Superko. The combined message from the arteriographic trials is that a more aggressive approach to lowering LDL cholesterol for secondary protection is justified. Despite LDL values that might not place patients with documented CAD at risk, it is likely that these patients too will benefit from aggressive lipoprotein manipulation.

detailed analysis essential

Much work remains to be done to define precisely which patient subgroups will benefit most from which single or combination therapy, say the researchers. It is no longer adequate to define improvements as change in total cholesterol, LDL, HDL or triglycerides, they say. Detailed lipoprotein analysis can enhance the clinician's ability to identify correctly lipoprotein abnormality and prescribe the correct therapy that will be physiologically and economically effective. Dr Superko recommends LDL and Lp(a) phenotype determination, methionine load testing, subclass distribution for **HDL2b** (the subclass associated with most improvements), Apo E isoform characterisation, Apo B and fibrinogen determination. The cost of wide-ranging diagnostic work-up is repaid in targeted treatment and reduced clinical events, he says.

2/9,K/5 (Item 1 from file: 148) [Links](#)

Gale Group Trade & Industry DB

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08653755 **Supplier Number:** 18275566 (THIS IS THE FULL TEXT )

**Berkeley HeartLab, Inc. Gains Exclusive License to New Heart Disease Diagnostic Test Panel from Ernest Orlando Lawrence Berkeley National Laboratory; New Diagnostic Test Panel Identifies Key Risk Factors Cited by American College of Cardiology.**

Business Wire , p5131204

May 13 , 1996

**Language:** English

**Record Type:** Fulltext

**Word Count:** 1687 **Line Count:** 00144

**Text:**

SAN MATEO, Calif.--(BUSINESS WIRE)--May 13, 1996--Berkeley HeartLab, Inc., a company focusing on new diagnostic technology for detecting heart disease, announced today it has signed a \$3.8 million, five-year research agreement with a technology option with Ernest Orlando Lawrence Berkeley National Laboratory (LBNL) to acquire exclusive worldwide rights to a new diagnostic technology that is a major improvement over existing cholesterol tests in measuring heart disease risk factors.

Cardiovascular disease is the nation's leading cause of death in the U.S., accounting for 35 to 40 percent of all deaths.

The proprietary screening technology is the foundation for a new diagnostic test panel being developed by the newly formed Berkeley HeartLab, a privately-held company that will commercialize the test panel, making it widely available to the public for the first time. The new technology is seen as the next generation in heart disease screening because it can identify the large number of heart disease candidates who have the same cholesterol profile as those who do not develop heart disease. Early screening for heart disease risk will assist physicians in better selecting appropriate therapy. In many cases, customized therapy will halt progression of coronary artery disease and therefore reduce the need for expensive surgery.

Measures Risk Factors Cited by ACC

Berkeley HeartLab also said the new heart disease test panel measures several newly discovered risk factors and abnormalities, including the size of low-density lipoprotein particles (LDL) -- often referred to as "bad cholesterol." The new abnormalities are among the major identifiable and manageable heart disease risk factors cited by the American College of Cardiology in a task force statement published in the association's April 1996 issue of the Journal of the American College of Cardiology. As a result, Berkeley HeartLab expects that cardiologists will begin to use the new test panel as a "first-line" screening for heart disease patients and family members.

Next Generation of Cholesterol Testing

Cardiovascular disease ranks as the leading cause of mortality and morbidity in the United States, causing 954,000 deaths in 1993, according to the American Heart Association. Of this total, more than 489,000 deaths

were due to coronary artery disease in the United States. Current testing methods used to determine an individual's risk of developing heart disease are generally confined to measuring total cholesterol, LDL, high-density lipoproteins (HDL or "good cholesterol") and triglycerides. Risk factors identified by these measurements include elevated cholesterol and elevated triglycerides, elevated LDL levels and low HDL levels. But these tests do not identify a substantial portion of patients who eventually develop heart disease.

#### Measuring New High-Risk Factors

A research team led by Ronald Krauss, M.D., director of the Department of Molecular and Nuclear Medicine at LBNL and H. Robert Superko, M.D., director of the Cholesterol, Genetics and Heart Disease Institute has developed a 10-test investigative panel which includes current parameters of cardiovascular risk -- total cholesterol, LDL, HDL and triglycerides -- plus six new tests designed to identify high-risk factors.

The 10-test panel has been used by LBNL researchers to investigate thousands of patients since 1990, as part of the lab's clinical research program. The licensing of the technology will allow the test to be made widely available commercially. Berkeley HeartLab said it plans to seek 510K approval for the investigative panel from the U.S. Food and Drug Administration (F.D.A.)

In addition to measuring current cardiovascular risk factors, the new diagnostic technology provide six other risk factor tests, including LDL particle size:

LDL Particle Size -- Low Density Lipoprotein, or LDL, particles contribute to heart disease risk and high LDL-cholesterol is well established as a heart disease risk factor. However, most people with heart disease do NOT have classic elevated LDL cholesterol. LDL particles can be present in a variety of sizes and the small LDL's (known as LDL pattern B) create substantially higher heart disease risk compared to large LDL's (known as LDL pattern A) even when LDL cholesterol is "normal."

Measurement of LDL cholesterol does not determine these two categories of LDL. The small LDL characteristic is determined, in any given individual, by inherited traits. Approximately 50 percent of men with heart disease express the small LDL trait and approximately 50 percent of their first degree relatives can express it as well. The presence of small LDL increases the risk of heart disease by 300 percent.

Patients with LDL pattern B can be successfully treated, often with a combination of diet, weight control, and lipid lowering medications. Diet and drug therapy recommendations are different for LDL pattern A individuals. Patients with LDL pattern B can respond quite well to specialized lipid lowering therapy and achieve heart disease stability, and some degree of regression.

HDL Subclassification -- HDL participated in "reverse cholesterol transport." An abundance of **HDL2b** reflects good "reverse cholesterol transport," while a deficiency of **HDL2b** reflects poor "reverse cholesterol transport." HDL cholesterol does not reflect the proportion of the different types of HDL's. Low **HDL2b** can be improved by specific therapy.

Lp(a) - Lp(a) is an LDL particle with the protein (a) attached. This inherited trait is present in approximately 33 percent of heart disease patients and in 50 percent of their first degree relatives. It increases

the risk of heart disease 300 percent and is not detected on routine blood tests. It is a powerful predictor of heart attacks in "young" men, and of vein graft blockage following bypass surgery. The presence of Lp(a) greatly increases the danger of other risk factors.

Apoprotein B - Apoprotein B is a single protein attached to the LDL particle. This test gives a more accurate measurement of the relative number of LDL particles than the current standard cholesterol blood test. Elevated apoprotein B identifies a high risk state even in the presence of "normal" LDL cholesterol. Elevated apoprotein B can be reduced with therapy.

Apoprotein AI - Apoprotein AI is one of several proteins attached to the HDL particle. It may be a better predictor of heart disease risk than HDL cholesterol. Low level of apoprotein AI can be increased with several therapies.

Apoprotein E Isoforms - Apoprotein E is attached to several lipoprotein classes and differences (isoforms) exist in "normal" and "abnormal" forms which are inherited. The presence of abnormal forms can predispose individuals to heart disease and blood lipid abnormalities. The presence, or absence, of different isoforms affects the potential success of diet and drug therapy.

The addition of these new tests provides physicians with more relevant diagnostic information for identifying heart disease candidates and assessing subsequent treatment. Early screening for and management of heart disease will allow physicians to more accurately design a customized treatment approach, reducing the need for expensive hospitalization or surgical intervention.

"The commercialization of this technology allows us to bring immediate benefits to millions of Americans who now suffer from cardiovascular disease," said Robert L. Swift, Ph.D., president and chief executive officer of Berkeley HeartLab. "More importantly, we will be able to identify the millions of Americans who have an inherited cardiovascular risk factors, so that the disease does not progress to a stage requiring costly surgical treatment."

Swift said Berkeley HeartLab will operate a centralized diagnostic service laboratory in Berkeley, Calif., that will analyze patient blood samples provided by cardiologists and a range of other physicians. The laboratory is certified under the F.D.A.'s CLIA (Clinical Laboratory Improvement Amendments) regulations. The commercial testing service is expected to begin operation in May 1996.

Researchers at the LBNL, who first isolated lipoproteins in 1949, have been world leaders in studying the connections between cholesterol and heart disease for more than 40 years. The LBNL was also the first to identify subclasses of lipoproteins and to determine that the ratio of high density-to-low density lipoproteins is a strong indicator of heart disease. The development of the testing technology and the licensing of it to the private sector is part of the laboratory's charter to convert its research breakthroughs into viable technologies for civilian applications.

First Product from New Company

The technology licensed from LBNL is the basis of the first product to be marketed by the newly formed Berkeley HeartLab, Inc., a privately-held company founded in March 1996. The company has raised an initial round of financing through individual investors. Berkeley HeartLab is headed by a

management team that includes co-founder Robert L. Swift, Ph.D., president and chief executive officer, co-founder Dennis J. Sheehan, M.D., a member of the board of directors, and David L. Kaufman, M.D., vice president, technical operations. H. Robert Superko, an internationally known lipid specialist and director of the Cholesterol, Genetics and Heart Disease Institute, serves as medical advisor to the company.

Robert L. Swift, Ph.D., co-founder, president, chief executive officer and director, was a co-founder and former vice president of operations and development of COR Therapeutics. Prior to joining COR, Dr. Swift was director, clinical research at Genentech and director, product planning and development at Pfizer Pharmaceuticals.

Dennis J. Sheehan, M.D., co-founder and a member of the board of directors, is a fellow of the American College of Cardiology and the American Society of Angiography. He is a cardiology consultant at Sequoia Hospital, Redwood City, CA, and a clinical assistant professor of medicine at Stanford University Medical Center.

David L. Kaufman, M.D., M.P.H., vice president, technical operations, was previously vice president - clinical affairs and medical director, Circadian, Inc., where he developed and managed disease management programs and participated in development and manufacture of diagnostic and electrosurgical devices.

CONTACT: Berkeley HeartLab, Inc.

Robert Swift, 415/378-8513

or

StratiPoint Group, Inc.

Mike Jackman or Carole Melis, 415/326-0420

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**Company Names:** Berkeley HeartLab Inc.--Licenses

**Industry Codes/Names:** BUS Business, General

**Descriptors:** Pharmaceutical industry--Licenses

**Product/Industry Names:** 8000251 (Heart & Cardiovascular R&D)

**Product/Industry Names:** 8730 Research and Testing Services

**File Segment:** NW File 649

2/3,K/3 (Item 1 from file: 813) [Links](#)

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0280713

NY038

**RESEARCH HIGHLIGHTS COMPLEXITIES OF CHOLESTEROL REMOVAL**

**Date:** June 25, 1990      **12:02 EDT**      **Word Count:** 533

**Correction:**

...their role in the uptake and transport of cholesterol examined.

Among HDL's subfractions are **HDL2B**, 2A, 3A, 3B, and 3C, with 2B being the largest and 3C the smallest. Scientists...



#### IV. Text Search Results from Dialog (Abstract dbs)

##### A. Abstract Databases -- Patent

[File 347] **JAPIO** Dec 1976-2008/Oct(Updated 090220)  
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[File 350] **Derwent WPIX** 1963-2009/UD=200919  
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Set	Items	Description
S1	10	S HDL()2B OR HDL2B OR (HIGH(2W)LIPOPROTEIN?)(2N)(2B OR 2()B) OR H()D()L() (2()B OR 2B) OR HDL2()B
S2	0	S S1 NOT AY>1999

## B. Abstract Databases – NON-PATENT

[File 35] **Dissertation Abs Online** 1861-2009/Mar  
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[File 583] **Gale Group Globalbase(TM)** 1986-2002/Dec 13  
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[File 474] **New York Times Abs** 1969-2009/Apr 13  
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[File 99] **Wilson Appl. Sci & Tech Abs** 1983-2009/Feb  
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[File 256] **TecInfoSource** 82-2009/Dec  
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Set	Items	Description
S1	2	S HDL() 2B OR HDL2B OR (HIGH(2W) LIPOPROTEIN?) (2N) (2B OR 2()B) OR H()D()L() (2()B OR 2B) OR HDL2()B
S2	1	S S1 NOT PY>1999

[File 5] **Biosis Previews(R)** 1926-2009/Apr W1  
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[File 155] **MEDLINE(R)** 1950-2009/Apr 13  
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[File 34] **SciSearch(R) Cited Ref Sci** 1990-2009/Apr W1  
(c) 2009 The Thomson Corp. All rights reserved.

[File 434] **SciSearch(R) Cited Ref Sci** 1974-1989/Dec  
(c) 2006 The Thomson Corp. All rights reserved.

[File 74] **Int.Pharm.Abs** 1970-2009/Jan B1  
(c) 2009 The Thomson Corporation. All rights reserved.

[File 42] **Pharm. News Index** 1974-2009/Mar W3  
(c) 2009 ProQuest Info&Learning. All rights reserved.

Set	Items	Description
S1	708	S HDL()2B OR HDL2B OR (HIGH(2W)LIPOPROTEIN?) (2N) (2B OR 2()B) OR H()D()L() (2()B OR 2B) OR HDL2()B
S2	534	S S1 NOT PY>1999
S3	343	S DEVICE? ? OR SYSTEM? ? OR APPARATUS?? OR TOOL? ? OR INDICATOR? OR INSTRUMENT? OR TEST? ? OR DIAGNOS? OR ENGINE? ? OR PROGNO? PROFIL? OR MARKER? OR PRODUCT? ? OR SERVICE? ? OR MEDICAL()LAB? OR PROBE OR PROBES OR RISK? OR PREDICT? OR FORECAST? OR PARAMETER? OR TRAIT? ? OR MEASUR? OR QUANTIF? OR ESTABLISH?
S4	62	S S2(12N)S3
S5	30	RD (unique items)
S6	0	S S2(30N) (COMMERCIAL OR LAB?()TEST? ?)
S7	0	S S2(30N) (DIAGNOS?() (ENGINE? ? OR TOOL? ? OR DEVICE? OR TEST? ?))
S8	0	S S2(30N) (LAB?(2N) (ENGINE? ? OR TOOL? ? OR DEVICE? OR TEST? ?))

5/5,K/11 (Item 11 from file: 5) [Links](#)

Fulltext available through: [STIC Full Text Retrieval Options](#)

Biosis Previews(R)

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11380149 **Biosis No.:** 199294081990

# **REDUCED HDL-2 CHOLESTEROL SUBSPECIES AND ELEVATED POSTHEPARIN HEPATIC LIPASE ACTIVITY IN OLDER MEN WITH ABDOMINAL OBESITY AND ASYMPTOMATIC MYOCARDIAL ISCHEMIA**

**Author:** KATZEL L I (Reprint); COON P J; BUSBY M J; GOTTLIEB S O; KRAUSS R M; GOLDBERG A P

**Author Address:** BALTIMORE VA MED CENT, GERIATRICS SERV, 3900 LOCH RAVEN BOULEVARD, BALTIMORE, MD 21218, USA\*\*USA

**Journal:** Arteriosclerosis and Thrombosis 12 ( 7 ): p 814-823 1992

**ISSN:** 1049-8834

**Document Type:** Article

**Record Type:** Abstract

**Language:** ENGLISH

**Abstract:** Silent myocardial ischemia (SI), an asymptomatic manifestation of coronary artery disease (CAD), was identified in 10% of apparently healthy nonsmoking, nondiabetic older (60  $\pm$  7 years, mean  $\pm$  SD) men with normal plasma cholesterol levels. We hypothesized that in the absence of other major risk factors for CAD, the men with SI would have reduced plasma levels of high density lipoprotein (HDL) and HDL2 subspecies due to an upper-body fat distribution (waist-to-hip ratio [WHR]), hyperinsulinemia, and abnormal postheparin plasma lipoprotein lipase (LPL) and hepatic lipase (HL) activities. Compared with 47 normal control subjects of similar age, obesity, and maximal aerobic capacity, the 18 men with SI had higher plasma triglyceride (TG) (162  $\pm$  71 versus 102  $\pm$  39 mg/dl,  $p$  < 0.001) and lower HDL-C (33  $\pm$  6 versus 37  $\pm$  7 mg/dl,  $p$  < 0.02) levels with no difference in low density lipoprotein cholesterol level. The **HDL2b** and HDL2a subspecies **measured** by gradient gel electrophoresis were also lower in the men with SI ( $p$  < 0.01). The plasma glucose and insulin responses during an oral glucose tolerance test were the same

in both groups. Postheparin plasma HL activity was significantly higher in 12 men with SI than in 41 control subjects (34  $\pm$  8 versus 27  $\pm$  10  $\mu$ mol/ml  $\cdot$  hr<sup>-1</sup>,  $p < 0.03$ ) and was correlated with log insulin area ( $r = 0.36$ ,  $p < 0.05$ ) and WHR ( $r = 0.32$ ,  $p < 0.05$ ) in the control subjects but not in the men with SI. In the control group, the percent HDL2b subspecies was correlated inversely with postheparin plasma HL activity ( $r = -0.46$ ,  $p < 0.01$ ,  $n = 41$ ) as well as WHR ( $r = -0.49$ ,  $p < 0.001$ ,  $n = 47$ ) and log insulin area ( $r = -0.37$ ,  $p < 0.05$ ,  $n = 47$ ) but not in the men with SI. Postheparin LPL activity was the same in both groups of men and did not correlate with HDL, WHR, insulin, or plasma TG levels. As the control subjects and men with SI had comparable degrees of abdominal obesity and hyperinsulinemia, these results suggest that the reduced HDL-C levels in men with SI may be related to elevations in HL activity. Thus, abdominal obesity, hyperinsulinemia, elevated TG levels, and low HDL-C and HDL2 subspecies level may predispose these older men to atherosclerosis.

**Registry Numbers:** 57-88-5: CHOLESTEROL; 9001-62-1: LIPASE

**Descriptors:** HIGH DENSITY LIPOPROTEIN ATHEROSCLEROSIS HYPERINSULINEMIA TRIGLYCERIDE LEVELS STATISTICS

**DESCRIPTORS:**

**Major Concepts:** Blood and Lymphatics--Transport and Circulation; Cardiovascular Medicine-- Human Medicine, Medical Sciences; Endocrine System--Chemical Coordination and Homeostasis; Enzymology-- Biochemistry and Molecular Biophysics; Geriatrics--Human Medicine, Medical Sciences; Nutrition; Physiology; Skeletal System--Movement and Support

**Biosystematic Names:** Hominidae--Primates, Mammalia, Vertebrata, Chordata, Animalia

**Common Taxonomic Terms:** Animals; Chordates; Humans; Mammals; Primates; Vertebrates

**Chemicals & Biochemicals:** CHOLESTEROL; LIPASE

**Concept Codes:**

04500 Mathematical biology and statistical methods  
 10064 Biochemistry studies - Proteins, peptides and amino acids  
 10066 Biochemistry studies - Lipids  
 10808 Enzymes - Physiological studies  
 11314 Chordate body regions - Abdomen  
 12002 Physiology - General  
 13203 Nutrition - Malnutrition and obesity  
 14506 Cardiovascular system - Heart pathology  
 14508 Cardiovascular system - Blood vessel pathology  
 15002 Blood - Blood and lymph studies  
 17008 Endocrine - Pancreas  
 18002 Bones, joints, fasciae, connective and adipose tissue - Anatomy  
 24500 Gerontology

**Biosystematic Codes:**

86215 Hominidae

**Abstract:** ...dl,  $p < 0.02$ ) levels with no difference in low density lipoprotein cholesterol level. The **HDL2b** and HDL2a subspecies **measured** by gradient gel electrophoresis were also lower in the men with SI ( $p < 0.01$ ...

5/5,K/25 (Item 6 from file: 73) [Links](#)

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 EMBASE

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0072270575 EMBASE No: 1982013193

**Differential use of apolipoproteins and lipoproteins as discriminators for an atherogenic risk**

DIFFERENTIALEINSATZ VON APOLIPOPROTEINEN UND LIPOPROTEINEN ALS  
DISKRIMINATOREN FUR EIN ATHEROGENES RISIKO

Mertz D.P.; Goehmann E.; Ostertag J.

Klin. am Park, 4934 Horn-Bad Meinberg, Germany

**Corresp. Author/Affil:** : Klin. am Park, 4934 Horn-Bad Meinberg, Germany

Medizinische Welt ( MED. WELT ) ( Germany ) December 1, 1981 , 32/43 (1611-1615)

**CODEN:** MEWEA **ISSN:** 0025-8512

**Document Type:** Journal ; Article **Record Type:** Abstract

**Language:** German

Apolipoproteins are now increasingly accepted as better indicators of atherosclerotic change than serum lipoproteins. Tests to verify this were carried out in 50 randomly selected patients in an ambulant practice. Lipid and lipoprotein values were collected; lipid values loose their predictive quality with age whereas lipoprotein value remains significantly associated with atherosclerotic change even in old age. As high density lipoproteins (HDL) represent a variety of substances consisting of various main and auxiliary components and because this composition can change during circulation it is suggested that **HDL2b** are more reliable in **predicting** any protective effect in serum. This however, can only be carried out by a few specialised laboratories; determination of the serum apolipoprotein concentration is a reliable indirect way of assessing HDL2a and 2b value as they appear to be narrowly connected.

**Drug Descriptors:**

\* apolipoprotein; \*lipoprotein

**Medical Descriptors:**

\* atherosclerosis; \*risk factor

cardiovascular system; major clinical study

**SECTION HEADINGS:**

Cardiovascular Diseases and Cardiovascular Surgery

Human Genetics

Clinical and Experimental Biochemistry

...and auxiliary components and because this composition can change during circulation it is suggested that **HDL2b** are more reliable in **predicting** any protective effect in serum. This however, can only be carried out by a few...

5/5,K/4 (Item 4 from file: 5) [Links](#)

Fulltext available through: [STIC Full Text Retrieval Options](#)

Biosis Previews(R)

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13223504 **Biosis No.:** 199698691337

**Plasma lipids: HDL subfraction and Lp(a) in a sample of centenarians**

**Author:** Barbagallo C M; Averna M (Reprint); Chessari S; Mangiacavallo G; Pollina R; Pupella E; Noto D; Marino G; Cefalu A B; Arces A; Frada G; Notarbartolo A

**Author Address:** Via A. De Saliba 14, 90145 Palermo, Italy\*\*Italy

**Journal:** Giornale della Arteriosclerosi 20 ( 1 ): p 47-53 1995 1995

**ISSN:** 0017-0224

**Document Type:** Article

**Record Type:** Abstract

**Language:** Italian

**Abstract:** Some previous reports associated the genetically determined presence of very high HDL levels with a prolonged life expectancy. Longevity is a condition characterized by absence or late onset of chronic diseases like atherosclerosis, which is strongly correlated with plasma lipids. In this investigation we examined lipid, apolipoprotein and Lp(a) levels and HDL subfractions distribution, determined by a polyacrilamide gradient gel electrophoresis in twenty-one subjects with an age of 100 years or more as a sample of longevity and in twenty-eight healthy normolipidemic subjects as controls. We did not find any significant difference in the mean levels of lipids and apolipoproteins between centenarians and controls: in particular both groups of subjects showed similar HDL-cholesterol concentrations (respectively 49.8+-14.5 mg/dl and 50.3+-12.1 mg/dl). Neither Lp(a) levels or prevalence of Lp(a) levels gt 30 mg/dl differed significantly between the two groups. When HDL subfractions were analysed, HDL-2b percent levels were significantly raised in centenarians in comparison with controls (30.2+-9.0% vs 24.5+-8.7%, p lt 0.03), while no difference was found for the other HDL subfractions. However we observed a large overlapping of individual values of HDL-2b between centenarians and controls. Since **HDL-2b** levels were found inversely correlated with CHD **risk**, we might speculate, in some cases, that they exert an influence also in healthy aging, even if we are very far from considering HDL subfraction distribution as a marker of longevity.

**Registry Numbers:** 142-54-1: LP(A); 57-88-5: CHOLESTEROL

**DESCRIPTORS:**

**Major Concepts:** Biochemistry and Molecular Biophysics; Blood and Lymphatics--Transport and Circulation; Cardiovascular System--Transport and Circulation; Geriatrics-- Human Medicine, Medical Sciences; Metabolism

**Biosystematic Names:** Hominidae--Primates, Mammalia, Vertebrata, Chordata, Animalia

**Organisms:** human (Hominidae)

**Common Taxonomic Terms:** Animals; Chordates; Humans; Mammals; Primates; Vertebrates

**Chemicals & Biochemicals:** LP(A); CHOLESTEROL

**Miscellaneous Terms: Concept Codes:** CHOLESTEROL; HIGH DENSITY LIPOPROTEIN SUBFRACTIONS; LIPID METABOLISM

**Concept Codes:**

10060 Biochemistry studies - General

13002 Metabolism - General metabolism and metabolic pathways

14501 Cardiovascular system - General and methods

15001 Blood - General and methods

24500 Gerontology

**Biosystematic Codes:**

86215 Hominidae

**Abstract:** ...observed a large overlapping of individual values of HDL-2b between centenarians and controls. Since **HDL-2b** levels were found inversely correlated with CHD **risk**, we might speculate, in some cases, that they exert an influence also in healthy aging...

5/5,K/3 (Item 3 from file: 5) [Links](#)

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Biosis Previews(R)

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14371952 **Biosis No.:** 199800166199

**Lipoprotein profile and high-density lipoproteins: Subfractions distribution in centenarians**

**Author:** Barbagallo Carlo M (Reprint); Averna Maurizio R; Frada Giovanni; Noto Davide; Cavera Giovanni; Notarbartolo Alberto

**Author Address:** Viale Francesco Scaduto 6/C, I-90144 Palermo, Italy\*\*Italy

**Journal:** Gerontology 44 ( 2 ): p 106-110 March-April, 1998 1998

**Medium:** print

**ISSN:** 0304-324X

**Document Type:** Article

**Record Type:** Abstract

**Language:** English

**Abstract:** In order to assess the role of HDL on longevity, we studied HDL subfraction distribution in centenarian women compared with a group of weight- and gender-matched healthy normolipidemic controls. We did not find any significant difference in the mean plasma lipid, apolipoprotein, and Lp(a) levels. On the contrary, in spite of similar HDL-cholesterol concentrations (1.32 +/- 0.41 mmol/l in centenarians vs. 1.32 +/- 0.25 mmol/l in controls, p = not significant), HDL2b and HDL3a levels were, respectively, significantly increased and significantly reduced in centenarians in comparison with controls (HDL2b 32.4 +/- 9.2% in centenarians vs. 23.4 +/- 7.7% in controls, p < 0.002, and Moreover, HDL2b levels were significantly raised and HDL3a levels were significantly reduced in centenarians in comparison with both 'middle-aged' and 'elderly' subjects, whereas no difference for any HDL subfraction was found between the two groups of controls of different ages. Age was significantly correlated with HDL2b and HDL3a (respectively, +0.452, p < 0.001, and -0.370, p < 0.01) in all subjects, but not with all the other lipid, lipoprotein and apolipoprotein **parameters**, but we observed a large overlapping of individual values of **HDL2b** between centenarians and controls. Since **HDL2b** levels were found to be inversely correlated with coronary heart disease **risk**, we could speculate that, in some cases, this may probably favor a healthy ageing, but long-term longitudinal studies are necessary to define the relative importance of HDL subfractions distribution as a marker of longevity. Probably other factors or clinical characteristics play a major role in the aging process.

**DESCRIPTORS:**

**Major Concepts:** Geriatrics--Human Medicine, Medical Sciences

**Biosystematic Names:** Hominidae--Primates, Mammalia, Vertebrata, Chordata, Animalia

**Organisms:** human (Hominidae)--female, centenarian, aged/80 and over

**Common Taxonomic Terms:** Animals; Chordates; Humans; Mammals; Primates; Vertebrates

**Chemicals & Biochemicals:** apolipoprotein--plasma level; high-density lipoprotein--subfraction distribution, plasma level; lipids--plasma level; lipoprotein a--plasma level

**Miscellaneous Terms: Concept Codes:** lipoprotein profile profile; longevity

**Concept Codes:**

24500 Gerontology

10064 Biochemistry studies - Proteins, peptides and amino acids

10066 Biochemistry studies - Lipids

**Biosystematic Codes:**

86215 Hominidae

**Abstract:** ...0.01) in all subjects, but not with all the other lipid, lipoprotein and apolipoprotein **parameters**, but we observed a large overlapping of individual values of **HDL2b** between centenarians and controls.

Since **HDL2b** levels were found to be inversely correlated with coronary heart disease **risk**, we could speculate that, in some cases, this may probably favor a healthy ageing, but...

5/5,K/17 (Item 17 from file: 5) [Links](#)

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Biosis Previews(R)

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08233214 **Biosis No.:** 198682079601

**LACK OF RELATIONSHIP IN HUMANS OF THE PARAMETERS OF BODY CHOLESTEROL METABOLISM WITH PLASMA LEVELS OF SUBFRACTIONS OF HIGH DENSITY LIPOPROTEIN OR LOW DENSITY LIPOPROTEIN OR WITH APOE ISOFORM PHENOTYPE**

**Author:** PALMER R H (Reprint); NICHOLS A V; DELL R B; RAMAKRISHNAN R; LINDGREN F T; GONG E L; BLUM C B; GOODMAN D S

**Author Address:** THE DEPARTMENT OF MEDICINE, COLUMBIA UNIVERSITY, COLLEGE OF PHYSICIANS AND SURGEONS, 6430 WEST 168TH STREET, NEW YORK, NY 10032, USA \*\*USA

**Journal:** Journal of Lipid Research 27 ( 6 ): p 637-644 1986

**ISSN:** 0022-2275

**Document Type:** Article

**Record Type:** Abstract

**Language:** ENGLISH

**Abstract:** The factors involved in regulating parameters of whole body cholesterol metabolism in humans have been explored in a series of investigations. Several physiological variables have been identified (weight, excess weight, plasma cholesterol, and age) that can predict 53-76% of the variation in production rate (PR) and in the sizes of the rapidly exchanging pool of body cholesterol (M1) and of the minimum estimates of the slowly exchanging pool of body cholesterol (M3min) and of total body cholesterol (Mtotmin). Surprisingly, measurements of the plasma levels of HDL cholesterol and of the major HDL apolipoproteins (apoA-I, A-II, and E) did not provide additional information useful in predicting parameters of whole body cholesterol metabolism. A study was therefore conducted to investigate possible relationships of the plasma levels of subfractions of lipoproteins, determined by analytic ultracentrifugation, and of apoprotein E phenotype, with the parameters of whole body cholesterol metabolism. Ultracentrifugal analysis of plasma lipoprotein subfractions was performed at the Donner Laboratory in 49 subjects; all of these subjects were currently undergoing whole body cholesterol turnover studies or had previously had such studies and were in a similar metabolic state as judged by plasma lipid and lipoprotein values. Apoprotein E phenotyping was carried out in 71 subjects. Differences in model parameters were sought among subjects with various apoprotein E phenotypes. Ultracentrifugal LDL subfractions **\*\*GRAPHIC\*\***. 0-2 (the region of Lp), **\*\*GRAPHIC\*\***. 0-7 (smaller LDL), **\*\*GRAPHIC\*\***. 7-12 (larger LDL), **\*\*GRAPHIC\*\***. 12-20 (IDL), and ultracentrifugal HDL subfractions **\*\*GRAPHIC\*\***. 0-1.5 (smaller HDL3), **\*\*GRAPHIC\*\***. 2-9 (larger HDL3 plus HDL2), and **\*\*GRAPHIC\*\***. 5-9 (larger HDL2 or **HDL2b**) were examined for correlations with each other and with **parameters** of whole body cholesterol metabolism. Although an interesting negative correlation between the smaller LDL subfraction **\*\*GRAPHIC\*\***. 0-7) and the HDL2-enriched subfraction **\*\*GRAPHIC\*\***. 2-9) was confirmed, neither the apoprotein E phenotype nor any of the analytic ultracentrifugal measurements (or their ratios) that were examined provided additional predictive information about parameters of whole body cholesterol metabolism. We conclude that these variables are not important determinants of parameters of whole body cholesterol metabolism in humans, and that other explanations must be sought for the remaining variability in these parameters.



**Registry Numbers:** 57-88-5: CHOLESTEROL

**DESCRIPTORS:**

**Major Concepts:** Biochemistry and Molecular Biophysics; Blood and Lymphatics--Transport and Circulation; Metabolism

**Biosystematic Names:** Hominidae--Primates, Mammalia, Vertebrata, Chordata, Animalia

**Common Taxonomic Terms:** Animals; Chordates; Humans; Mammals; Primates; Vertebrates

**Chemicals & Biochemicals:** CHOLESTEROL

**Concept Codes:**

10006 Clinical biochemistry - General methods and applications

10064 Biochemistry studies - Proteins, peptides and amino acids

10066 Biochemistry studies - Lipids

10504 Biophysics - Methods and techniques

13006 Metabolism - Lipids

13008 Metabolism - Sterols and steroids

15002 Blood - Blood and lymph studies

**Biosystematic Codes:**

86215 Hominidae

**Abstract:** ...HDL3), **\*\*GRAPHIC\*\***. 2-9 (larger HDL3 plus HDL2), and **\*\*GRAPHIC\*\***. 5-9 (larger HDL2 or **HDL2b**) were examined for correlations with each other and with **parameters** of whole body cholesterol metabolism. Although an interesting negative correlation between the smaller LDL subfraction...

5/5,K/16 (Item 16 from file: 5) [Links](#)

Biosis Previews(R)

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08646359 **Biosis No.:** 198784000508

**SEPARATION AND CHARACTERIZATION OF HIGH-DENSITY LIPOPROTEIN SUBPOPULATIONS BY GEL PERMEATION CHROMATOGRAPHY**

**Author:** CLIFTON P M (Reprint); MACKINNON A M; BARTER P J

**Author Address:** DEP MED BIOCHEMISTRY, FLINDERS MED CENT, BEDFORD PARK, 5042 SOUTH AUSTRALIA **\*\*AUSTRALIA**

**Journal:** Journal of Chromatography Biomedical Applications 414 ( 1 ): p 25-34 1987

**Document Type:** Article

**Record Type:** Abstract

**Language:** ENGLISH

**Abstract:** High density lipoproteins (HDL) contain at least five distinct subpopulations when analyzed by gradient gel electrophoresis. This report represents the first description of a simple technique for isolating these subpopulations of HDL in quantities sufficient to enable characterization in terms of particle size, apolipoprotein AII content and chemical composition. Lipoproteins were separated and subfractionated on a column of Superose 6B using a fast protein liquid chromatography **system**. Five normal subjects were studied: **HDL2b** and HDL3a were isolated as essentially single subpopulations from all subjects, while HDL2a could be isolated from only three of the subjects. HDL3b was isolated in a relatively impure form (70%) from all subjects. Identical subpopulations were identified in each subject by gradient gel electrophoresis of unseparated HDL.

**Descriptors:** HUMAN

**DESCRIPTORS:**

**Major Concepts:** Biochemistry and Molecular Biophysics; Blood and Lymphatics--Transport and Circulation; Clinical Chemistry--Allied Medical Sciences; Methods and Techniques

**Biosystematic Names:** Hominidae--Primates, Mammalia, Vertebrata, Chordata, Animalia

**Common Taxonomic Terms:** Animals; Chordates; Humans; Mammals; Primates; Vertebrates

**Concept Codes:**

10006 Clinical biochemistry - General methods and applications

10054 Biochemistry methods - Proteins, peptides and amino acids

10056 Biochemistry methods - Lipids

10064 Biochemistry studies - Proteins, peptides and amino acids

10066 Biochemistry studies - Lipids

10504 Biophysics - Methods and techniques

15001 Blood - General and methods

**Biosystematic Codes:**

86215 Hominidae

**Abstract:** ...separated and subfractionated on a column of Superose 6B using a fast protein liquid chromatography **system**. Five normal subjects were studied: **HDL2b** and HDL3a were isolated as essentially single subpopulations from all subjects, while HDL2a could be...

5/5,K/6 (Item 6 from file: 5) [Links](#)

Fulltext available through: [STIC Full Text Retrieval Options](#)

Biosis Previews(R)

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12443151 **Biosis No.:** 199497464436

**Structural and functional assessment of high-density lipoprotein heterogeneity**

**Author:** Dobiasova Milada (Reprint); Frohlich Jiri J

**Author Address:** Inst. Physiol., Acad. Sciences Czech Republic, Vidensak 1083, 142 20 Praha 4-Krc, Czech Republic\*\*Czech Republic

**Journal:** Clinical Chemistry 40 ( 8 ): p 1554-1558 1994 1994

**ISSN:** 0009-9147

**Document Type:** Article

**Record Type:** Abstract

**Language:** English

**Abstract:** We studied the heterogeneity of high-density lipoproteins (HDL) in plasma of 110 subjects, using three different methods: (a) gradient gel electrophoresis (GGE); (b) electroimmunoassay, to measure the concentration of lipoprotein particles containing apoprotein (apo) AI but no apoAII (LP AI); and (c) cholesterol esterification rate (FER-HDL) in very-low- and low-density lipoprotein-depleted plasma. There were two study groups: patients with hypertension, whose plasma lipid profile was similar to their respective controls, and patients with hypoalphalipoproteinemia (hypoalpha), whose family members served as controls. Values for FER-HDL were significantly higher in both risk groups than in their respective controls. LP AI was significantly decreased only in the hypoalpha subjects. Generally, LP AI and FER-HDL were inversely related. LP AI correlated strongly with plasma HDL-cholesterol, apo AI, and LP AI/AII; FER-HDL correlated with those values inversely. LP AI, but not FER-HDL, correlated with HDL free cholesterol. On the other hand, FER-HDL correlated strongly with plasma concentrations of triglycerides and with the plasma ratio of total/HDL-cholesterol while LP AI did not. GGE determination of the composition of HDL

subspecies showed that both FER-HDL and LP AI were significantly related to the content of HDL-2b particles; FERHDL inversely, LP AI directly; the relative amount of HDL-3b,c particles correlated only with FER-HDL. We conclude that GGE and FER-HDL can be used to **quantify** both the apparently protective (**HDL-2b**) and **risk**-associated (HDL-3b,c) particles, whereas the concentration of LP AI in plasma mainly reflects the concentration of the HDL-2 subpopulation.

**Registry Numbers:** 57-88-5: CHOLESTEROL; 9031-14-5: LECITHIN:CHOLESTEROL ACYLTRANSFERASE

**DESCRIPTORS:**

**Major Concepts:** Biochemistry and Molecular Biophysics; Cardiovascular Medicine--Human Medicine, Medical Sciences; Clinical Chemistry--Allied Medical Sciences; Enzymology--Biochemistry and Molecular Biophysics; Metabolism

**Biosystematic Names:** Hominidae--Primates, Mammalia, Vertebrata, Chordata, Animalia

**Organisms:** human (Hominidae)

**Common Taxonomic Terms:** Animals; Chordates; Humans; Mammals; Primates; Vertebrates

**Chemicals & Biochemicals:** CHOLESTEROL; LECITHIN:CHOLESTEROL ACYLTRANSFERASE

**Miscellaneous Terms: Concept Codes:** APOPROTEIN-AI; CHOLESTEROL ESTERIFICATION RATE; CLINICAL CHEMISTRY; HYPERTENSION; HYPOALPHALIPOPROTEINEMIA; LECITHIN:CHOLESTEROL ACYLTRANSFERASE

**Concept Codes:**

10006 Clinical biochemistry - General methods and applications  
10064 Biochemistry studies - Proteins, peptides and amino acids  
10066 Biochemistry studies - Lipids  
10067 Biochemistry studies - Sterols and steroids  
10506 Biophysics - Molecular properties and macromolecules  
10806 Enzymes - Chemical and physical  
10808 Enzymes - Physiological studies  
13006 Metabolism - Lipids  
13008 Metabolism - Sterols and steroids  
13020 Metabolism - Metabolic disorders  
14508 Cardiovascular system - Blood vessel pathology

**Biosystematic Codes:**

86215 Hominidae

**Abstract:** ...only with FER-HDL. We conclude that GGE and FER-HDL can be used to **quantify** both the apparently protective (**HDL-2b**) and **risk**-associated (HDL-3b,c) particles, whereas the concentration of LP AI in plasma mainly reflects...

?

2/5,K/1 (Item 1 from file: 35) [Links](#)

Dissertation Abs Online

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829412 ORDER NO: AAD83-28920

**THE INTERACTION OF PLASMA LIPOPROTEINS WITH PHOSPHATIDYLCHOLINE VESICLES (HDL)**

**Author:** HUNTER, JAMES ARTHUR

**Degree:** PH.D.

**Year:** 1983

**Corporate Source/Institution:** UNIVERSITY OF CALIFORNIA, BERKELEY ( 0028 )

**Source:** Volume 4408B of Dissertations Abstracts International.

PAGE 2313 . 221 PAGES

**Descriptors:** BIOPHYSICS, GENERAL

**Descriptor Codes:** 0786

In these studies, plasma lipoproteins were incubated with small unilamellar vesicles of phosphatidylcholine, and the interaction products were characterized in terms of chemical composition and physical-chemical properties.

Separate interactions (37(DEGREES)C) of all density subclasses of high density lipoproteins (HDL) with vesicles resulted in the formation of three products: liposomes with associated apolipoproteins, discoidal complexes of phospholipids and apolipoproteins, and lipoproteins with an altered chemical composition. The lipoprotein product resulted from phospholipid uptake by and apolipoprotein dissociation from the density subclasses of HDL (HDL(,3), HDL(,2a), and **HDL(,2b)**). The increase in phospholipid content of these subclasses was highly correlated with the decrease in apolipoprotein content.(,)

The major protein component of HDL, apolipoproteinA-I (apoA-I), was the primary apolipoprotein dissociated from the subclasses. Dissociation of apolipoprotein had not reached a limiting value for any subclass in the range of molar ratios (PC:HDL) investigated. At the highest molar ratios for the incubation mixtures, the average number of apoA-I molecules dissociated per HDL particle was 0.75:1, 1.2:1, and 2.1:1 for HDL(,3), HDL(,2a), and **HDL(,2b)**, respectively.

## **V. Additional Resources Searched**

No additional results of relevance found in the additional databases identified in the cover correspondence.